FEB 6 1931

Medical Lib.

VOLUME 22 NUMBER 2

ARCHIVES OF SURGERY

EDITORIAL BOARD

DEAN LEWIS, Baltimore

EVARTS A. GRAHAM, St. Louis WALLACE I. TERRY, San Francisco

HUGH CABOT, Rochester, Minn. WILLIAM DARRACH, New York

EDWARD STARR JUDD, Rochester, Minn.

FEBRUARY, 1931

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second-Class Matter, July 10, 1925, at the Postoffice at Chicago, Illinois, Under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of Oct. 3, 1917, authorized July 29, 1920.

CONTENTS OF PREVIOUS NUMBER

JANUARY, 1931. NUMBER 1

Peritonitis: I. The Correlation of the Bacteriology of the Peritoneal Exudate and the Clinical Course of the Disease in One Hundred and Six Cases of Peritonitis. Frank L. Meleney, M.D.; Harold D. Harvey, M.D., and Helen Zaytseff Jern, M.D., New York.

Oxygen Therapy: Insufflation into Oral Pharynx. A. J. Wineland, M.D., and Ralph M. Waters, M.D., Madison, Wis.

The Rôle of Toxin of Bacillus Welchii in the Toxemia of Acute Intestinal Obstruction: An Experimental Study. Herbert F. Thurston, M.D., Indianapolis.

Continuous Intravenous Injection of Dextrose in Ringer's Solution: Its Technic and Indications, and a New Intravenous Cannula. J. Shelton Horsley, M.D., and Guy W. Horsley, M.D., Richmond, Va.

The Fate of the Cortical Bone Graft. Charles Davison, M.D., and Adolph Kraft, M.D., Chicago.

Vaccination Against Peritonitis in Surgery of the Colon: Further Report. Fred W. Rankin, M.D., and J. Arnold Bargen, M.D., Rochester, Minn.

Ileocecal Cysts. Earle Drennen, M.D., Birmingham, Ala.

The Closed Method of Treatment of Fractures of the Ankle Joint. Carlo Lórenzetti, M.D., Milan, Italy.

The Regeneration of Hyaline Cartilage in Joints: An Experimental Study. A. R. Shands, Jr., M.D., Durham, N. C.

COPYRIGHT, 1931, BY THE AMERICAN MEDICAL ASSOCIATION

ARCHIVES OF SURGERY

VOLUME 22 FEBRUARY, 1931 NUMBER 2

DIABETIC GANGRENE

MEDICAL TREATMENT AND PROPHYLAXIS *

HOWARD F. ROOT, M.D.

BOSTON

ETIOLOGY

Exclude infection and trauma, and death from diabetic gangrene would disappear. At the New England Deaconess Hospital in the service of Dr. E. P. Joslin and myself, from January, 1923, to January, 1930, thirty-one deaths occurred in one hundred and eighty cases of diabetic gangrene or infection of the feet requiring amputation of the toes or legs. In only seven were the causes of death not directly due to infection. The spontaneous occurrence of gangrene, trauma or infection is as rare in the diabetic as in nondiabetic persons of a similar age. It therefore seems clear that although premature arteriosclerosis, differing somewhat from nondiabetic arteriosclerosis, is present in the diabetic patient, the frequency as well as the high mortality of gangrene are dependent on a specific diabetic factor which for lack of a better term I must call lowered resistance to trauma and infection due to the metabolic disturbance peculiar to diabetes, perhaps especially manifested in the skin. Another point of contrast appears in the relative rarity of severe infection in cases of thrombo-angiitis obliterans in the nondiabetic person. Severe infections most commonly occur in patients with diabetes who have received inadequate treatment and whose condition therefore is not controlled. According to Campbell,[1] in animals that do not receive insulin suppuration often occurs after pancreatectomy, while in those given an adequate amount of insulin and food healing occurs by first intention. The paradoxic, yet I believe true, observation that diabetic patients with a given degree of infection or gangrene of the feet are on the whole somewhat better subjects for surgical treatment than nondiabetic patients is probably due to the fact that this specific diabetic factor can be brought under control by adequate medical treatment. It follows, therefore, that control of diabetes by appropriate treatment is of primary importance in the prophylaxis of gangrene and as the foundation for successful surgical treatment whether operative or nonoperative.

* Submitted for publication, May 3, 1930.

1. Campbell, quoted by Joslin: Treatment of Diabetes Mellitus, ed. 4, Philadelphia, Lea & Febiger, 1928, p. 770.

PATHOLOGY

The impairment of the blood supply to the feet by vascular disease is always a factor in diabetic gangrene. In legs amputated for gangrene this arteriosclerosis is characterized by the fact that although the arteries of the leg are of the muscular type, the changes found are predominately of the type which in nondiabetic patients is found in the elastic arteries, such as the aorta. In the legs of nondiabetic patients the changes consisting of calcification, necrosis and sclerosis, occurring chiefly in the media, form the so-called Mönckeberg type of arteriosclerosis. No great narrowing of the lumen occurs until, through degeneration of the intima, a suddenly formed thrombus completely occludes the artery. Typical senile gangrene, usually of the dry type, then develops. Such senile changes may be present in the arteries of persons developing diabetes in the late middle life, and hence senile arteriosclerotic gangrene may occur in a diabetic person. Characteristically, however, in the arteries of diabetic persons there is found marked intimal involvement [2] consisting of a heaping up of intima with deposition of fatty material in which many cholesterol crystals are seen. The heaping up of intima results in an early and gradual narrowing of the lumen with a limitation in the amount of blood supply. This process is usually of slow development, but may progress fairly rapidly in the course of a few months. As a result of this slowly progressive occlusion of the lumen, there tends to develop a compensatory collateral circulation, so that in time in a foot in which the large arteries are partially or completely obstructed the blood supply may still be sufficient to keep the foot alive and in apparently satisfactory condition for the ordinary burdens of life. Gangrene develops when a sudden occlusion or thrombosis occurs for which the foot is not adequately prepared, or more frequently when the edema and inflammation, due to trauma or infection, cause thrombosis of small arteries. The infection spreads rapidly, due to the lack of resistance in the diabetic patient, and gangrene follows. Owing in part to the collateral circulation, diabetic gangrene is usually moist, and the foot frequently warm.

Calcification in the vessels of diabetic persons tends to develop at any age from youth upward and is favored by two factors: increasing age and increasing duration of diabetes. It occurs in other vessels, notably the aorta and coronary arteries; in many cases hypertension associated with arteriolar sclerosis in the kidneys, eyes and heart contributes to the final pathologic picture. The organ in which arteriosclerosis threatens life most frequently is the heart. In eighty autopsies at the Deaconess Hospital every patient over the age of 40 with diabetes of five years' duration had had sclerosis of the coronary arteries. The chief cause of death of patients with diabetic gangrene, with the excep-

2. Root: New York State J. Med. **28**:1287, 1928.

tion of septicemia developing at the time of the gangrene, has been cardiovascular disease. Coronary thrombosis with infarction of the heart, recent or healed, occurred in 12 per cent of two hundred and eighty-two diabetic persons whose autopsies were studied by Warren [3] and in 25 per cent of one hundred cases at the Deaconess Hospital. In treating patients who have diabetic gangrene, therefore, one deals with persons who have generalized arteriosclerosis, the extent and degree of which vary with the duration of the diabetes and the age of the patient; if the diabetes is uncontrolled, there is also a disturbance of metabolism which in some way reduces the resistance to infection. Yet the nature of the change in larger arteries affected by diabetes gives opportunity for an extensive development of a protective collateral circulation, which, barring trauma and infection, in time may become sufficient to prevent gangrene.

INCIDENCE OF GANGRENE

In the last four years, one in eleven diabetic persons over the age of 50 died of it as compared with one in eight dying during the preceding four years. The increasing longevity of diabetic patients brings more youthful persons into the arteriosclerotic zone. The average age of thirty-four persons with fatal diabetes in the hospital during 1929 was 63.5 years. The patients with gangrene are in reality much older than their chronological age, if one considers their extensive vascular disease and the duration of life after gangrene. Indeed, in the past it almost seems as if a year of diabetes added a year to a patient's true age. It should not do so in the future.

PREVENTION OF GANGRENE

The immediate causes of gangrene are preventable. On March 31, 1930, of seven patients in the New England Deaconess Hospital who had suffered amputations of legs or toes, three had cut corns without precautions. Clean hands and clean feet would have saved their legs. The fourth patient stepped on a nail and continued to work in spite of suppuration. The fifth and sixth had blisters produced by tight shoes. The seventh walked barefoot in zero weather, striking his toe without remembering any injury, so deficient in sensation or so slight was the trauma that resulted in gangrene. Defective vision resulting in too deep cutting of corns, improper shoes causing trauma from pressure, unprotected feet and neglect of minor infections are causes that may be combated by the education of patients and by careful but energetic treatment by physicians.

The feet of diabetic persons are vulnerable because they are mechanically deformed. The feet of diabetic patients over 50 years of age are rarely free from obvious mechanical handicaps. Hammer

3. Warren: Pathology of Diabetes, Philadelphia, Lea & Febiger, 1930, p. 146.

toes and toes made stiff by arthritic changes cause abnormal points of pressure, with the formation of calluses; these can be kept free from infection only if they are given proper care. Bunions, with displacement of the great toes, not only produce abnormal points of pressure over the metacarpophalangeal joint, but often cause soft corns on the inner aspect of the fourth toes and calluses on the lateral and under surfaces of the first toes, so that every bunion has at least three potentional points for infection. Flat anterior arches result in calluses over the heads of the metatarsals, and the abnormal pressure is in most cases the chief predisposing factor in producing perforating ulcer of the sole. Deformities of the small toes due to long-continued wearing of ill-fitting shoes result in the formation of soft corns on the lateral surfaces of the toes at those points underlying the small phalangeal joints. When these soft corns become infected, involvement of the joints is almost certain if healing has not occurred within two weeks. Loss of flexibility of the ankle joint should not be forgotten as a factor in the production of a stiff foot and therefore as a cause of pressure calluses.

The presence of chronic infections about the foot is of importance. Epidermophytosis is present in 70 per cent of diabetic patients; although not dangerous in itself, the lesions produced by this infection provide a portal of entry for a pyogenic organism with resultant osteomyelitis, lymphangitis or gangrene. The surfaces between the toes should always be examined for the dead white macerated skin or soft corns due to epidermophytosis. A large number of diabetic patients harbor mild infection in or about calluses or corns, which at times flares up and may give rise to lymphangitis or even gangrene.

Finally, gangrene is precipitated by faulty care of vulnerable feet by patients infirm on account of age and handicapped by poor vision and defective sensation in the feet. A corn is cut and blood drawn when the feet or the hands are not clean. A blister is picked or a pressure sore is produced by the wearing of shoes with a torn lining or a new shoe before the stiffness has gone out of it. The skin of a diabetic person is delicate. One can break it by rubbing with a turkish towel too vigorously, and if the broken area is not protected, a few days later the patient may have not only infection but gangrene. Shoes that are long enough and soft enough should be worn; also proper hose that are not too tight or so large that wrinkling will cause blisters or sores when worn. Medium weight stockings of wool and cotton should be worn and changed daily.

Most important of all is the education of every diabetic patient in the care of his feet. No patient leaves the New England Deaconess Hospital without a demonstration in the Beauty Parlor for Diabetic Feet of the proper care of the feet, and the following instructions are given for use at home:

Hygiene of the Feet.—1. Wash the feet daily with soap and water. Dry thoroughly, especially between the toes, using pressure rather than vigorous rubbing.

2. When thoroughly dry, rub well with hydrous wool fat as often as necessary to keep the skin soft, supple and free from scales and dryness, but never render the feet tender.

3. If the feet become too soft, rub once a day with alcohol.

4. If the nails are brittle and dry, soften by soaking in warm water one-half hour each night and apply hydrous wool fat generously under and about the nails, and bandage loosely. Clean the nails with orange-wood sticks. Cut the nails only in a good light and after a bath, when the feet are very clean. Cut the nails straight across to avoid injury to the toes. If you go to a chiropodist, tell him you have diabetes.

5. Wear shoes of soft leather that fit and are not tight (neither narrow nor short). Wear new shoes one-half hour only on the first day and increase the period one hour daily.

6. Do not step on floor with bare feet.

7. Use bed socks instead of hot water bottles, bags or electric heaters.

Treatment of Corns and Callosities.—1. Wear shoes that fit and cause no pressure.

2. Soak the foot in warm, not hot, soapy water. Rub off with gauze or file off the dead skin on or about callus or corn. Do not tear it off. A corn may be painted with the following mixture: salicylic acid, 1 drachm; collodion, 1 ounce. Repeat for four nights; then, after soaking in warm water, the corn will come off easily. If it does not come off easily without bleeding, repeat the treatment for four nights.

3. Do not cut corns or callosities.

4. Prevent calluses under the ball of the foot,

(*a*) By exercises such as curling and stretching the toes twenty times a day.

(*b*) By finishing each step on the toes and not on the ball of the foot.

Aids in Treatment of Imperfect Circulation.—Cold Feet: 1. Exercises. Bend the foot down and up as far as it will go six times. Describe a circle to the left with the foot six times and then to the right. Repeat morning, noon and night.

2. Massage with hydrous wool fat or cocoa butter.

3. Do not wear circular garters. Do not sit with the knees crossed.

Treatment of Abrasions of the Skin.—1. Proper first-aid treatment is of the utmost importance even in apparently minor injuries. Consult your physician immediately.

2. Avoid strong irritating antiseptics, such as sulphonaphthol and iodine.

3. As soon as possible after injury certain surgeons recommend the application of sterile gauze saturated with medicated alcohol or hexylresorcinol (S.T. 37). Keep wet for one hour by pouring on more alcohol or hexylresorcinol. Sterile gauze in sealed packets may be purchased at drug stores.

4. Elevate, and as much as possible until recovery, avoid using the foot.

5. Consult your physician for any redness, pain, swelling or other evidence of inflammation.

MEDICAL TREATMENT

Dietary.—Prompt and aggressive treatment aimed to improve the carbohydrate tolerance so that the patient can utilize a diet more nearly normal is needed. The patient with gangrene always has mild

TABLE 1.—*Data on Sixteen Patients Treated During 1929*

Case Number	Sex	Age	Weight, Lbs.	Wassermann	Duration: Diabetes, Years	Duration: Gangrene, Weeks	Operations (Same Leg—This Entry): Minor Amputations: Date	Minor Amputations: Type	Minor Amputations: Anesthesia	Major Amputations: Date	Major Amputations: Type	Major Amputations: Anesthesia	Days in Hospital	Complications: Angina	Complications: Nephritis: Nonprotein Nitrogen	Complications: Nephritis: Albumin	Complications: Nephritis: Blood Pressure	Complications: Miscellaneous
6607	F	51	123	Neg.	9	5-8	10/ 3/28	Amputation of 2 toes	Spinal	1/ 1/29	Amputation of lower part of leg	Spinal	115	0	39	vst	110/60	0
7784	M	68	160	Neg.	½	6	.....	0	.....	5/ 7/29	Amputation of lower part of leg	Spinal	21	0	27	cpt	122/76	0
7707	F	62	108	Neg.	5	4	.....	0	.....	2/ 9/29	Gritti-Stokes	Spinal	52	0	27	vst	140/80	0
6926	M	63	123	.....	20	4	5/11/29 5/23/29	Amputation of 2 toes Amputation of 1 toe	Spinal Spinal	6/ 4/29	Gritti-Stokes	Spinal	48	0	56	st	120/74	0
6896	F	60	159	Neg.	⅔	3-6	12/31/28	Amputation of 1 toe	Spinal	1/30/29	Gritti-Stokes	Spinal	60	0	..	vst	120/80	Auricular fibrillation
7652	M	62	...	Pos.	1/30+	3	.....	0	.....	2/14/29	Amputation of thigh	Spinal	31	0	..	vst	158/95	Tertiary syphilis and retinitis
7750	M	55	205	Neg.	⅛	8+	1/26/29 2/ 4/29 2/ 8/29	Amputation of toe and drainage Amputation of toe and drainage I. & D. foot	Spinal Spinal Spinal	2/13/29	Gritti-Stokes	Spinal	77	0	40	st	120/80	0

Case Number	Average Daily Diets	Carbohydrate	Protein	Fat	Calories	Insulin	B. S.
6607	Preoperative	109	65	98	1,596	5	0.29
	First week...	77	49	92	1,332	35	0.19
	Second week.	82	51	94	1,423	39	....
	Third week..	88	58	104	1,520	45	....
	Discharge....	96	66	115	1,683	25	0.12
7784	Preoperative	106	49	83	1,367	0	0.08
	First week...	101	45	73	1,241	0	0.12
	Second week.	134	55	94	1,602	0	0.13
	Third week..	150	56	98	1,706	0	....
	Discharge....	150	56	98	1,706	0	....
7707	Preoperative	106	57	88	1,442	25	0.19
	First week...	90	42	77	1,221	38	0.18
	Second week.	108	54	86	1,422	26	....
	Third week..	108	54	92	1,496	20	0.17
	Discharge....	108	54	93	1,485	10	0.16
6926	Preoperative	91	52	84	1,328	15	0.20
	First week...	86	29	58	982	31	....
	Second week.	97	50	79	1,299	20	0.18
	Third week..	110	55	82	1,398	18	....
	Discharge....	114	58	103	1,615	5	0.39
6896	Preoperative	104	51	65	1,205	10	0.20
	First week...	93	39	50	1.032	10	0.29
	Second week.	101	39	61	1,109	10	....
	Third week..	102	43	62	1,138	10	....
	Discharge....	108	44	61	1,157	10	....
7652	Preoperative	101	63	85	1,421	30	0.12
	First week...	102	65	108	1,640	31	0.17
	Second week.	106	66	113	1,705	20	0.18
	Third week..	106	66	113	1,705	20	....
	Discharge....	106	66	113	1,705	20	0.13
7750	Preoperative	100	65	98	1,542	75	0.25
	First week...	100	62	90	1,458	50	0.23
	Second week.	100	62	90	1,458	41	0.24
	Third week..	103	64	93	1,505	30	0.24
	Discharge....	110	75	102	1,658	5	0.16

7780	F	45	154	Neg.	14	4-6	.....	0	.....	12/22/28	Gritti-Stokes	Spinal	137	+	31	lt	180 — 100	Carbuncle of buttock	Preoperative	89	37	70	1,124	20	0.25
																			First week...	85	49	79	1,247	45	0.21
																			Second week.	98	51	83	1,343	35	0.14
																			Third week..	108	53	87	1,427	27	0.14
																			Discharge....	122	64	122	1,842	0	0.14
3364	M	67	206	.....	6	1-2	.....	0	.....	4/13/29	Gritti-Stokes	Spinal	57	0	44	st	120 — 80	Chronic pulmonary tubercu-losis	Preoperative	104	48	72	1,256	30	0.24
																			First week...	87	33	78	1,182	47	0.23
																			Second week.	87	38	84	1,256	38	0.16
																			Third week..	95	50	81	1,309	27	0.20
																			Discharge....	112	59	20	1,404	10	0.12
7800	F	66	...	Neg.	1/26	2	.....	0	.....	5/13/29	Gritti-Stokes	Spinal	78	0	31	vst	175 — 115	0	Preoperative	59	36	55	875	0	0.18
																			First week...	53	8	15	379	17	0.33
																			Second week.	72	35	44	824	18	0.15
																			Third week..	74	27	58	926	10	0.17
																			Discharge....	150	65	86	1,634	0	0.16
6575	M	49	166	Neg.	0	3-4	.....	0	.....	1/ 2/29	Amputation of lower part of leg	Spinal	103	0	..	vst	132 — 84	0	Preoperative	152	73	118	1,962	0	0.21
																			First week...	120	57	116	1,752	2	....
																			Second week.	152	73	118	1,962	10	....
																			Third week..	152	73	118	1,962	5	0.11
																			Discharge....	162	76	118	2,026	0	0.15
7884	F	59	118	Neg.	7	3	.....	0	.....	4/ 1/29	Gritti-Stokes	Spinal	58	0	32	vst	140 — 80	0	Preoperative	95	55	80	1,320	50	0.29
																			First week...	97	54	80	1,324	61	0.20
																			Second week.	96	50	80	1,304	41	0.20
																			Third week..	97	49	80	1,304	20	0.20
																			Discharge....	89	50	92	1,384	0	0.18
5619	F	50	124	Neg.	9	3	.....	0	.....	3/ 7/29	Gritti-Stokes	Spinal	40	0	..	spt	130 — 80	0	Preoperative	94	55	88	1,388	27	0.22
																			First week...	88	33	65	1,069	38	....
																			Second week.	91	43	81	1,265	34	0.24
																			Third week..	93	54	88	1,380	28	....
																			Discharge....	89	52	88	1,356	20	....
8005	F	68	108	Neg.	5	8-10	.....	0	.....	5/21/29	Amputation of thigh	Spinal	53	0	47	spt	160 — 74	0	Preoperative	92	48	81	1,280	0	0.18
																			First week...	97	42	72	1,208	15	0.16
																			Second week.	115	55	83	1,427	21	0.12
																			Third week..	121	64	90	1,550	12	....
																			Discharge....	144	51	84	1,536	0	0.19
8011	F	78	165	Neg.	6	6	.....	0	.....	5/29/29	Amputation of thigh	Spinal	40	+	33	spt	136 — 54	Auricular fibrillation	Preoperative	84	39	88	1,284	0	0.22
																			First week...	81	33	69	1,077	23	0.22
																			Second week.	102	43	94	1,426	21	0.17
																			Third week..	95	48	87	1,355	10	....
																			Discharge....	104	50	92	1,444	10	0.14
8264	F	66	...	Neg.	⅛	6-8	.....	0	.....	7/ 7/29	Amputation of thigh	Spinal and ethylene	78	+	35	spt	100 — 70	Acute coronary thrombosis at onset of angina pectoris	Preoperative (one meal)...	44	10	26	458	0	0.13
		—			—														First week...	96	26	52	956	0	0.09
																			Second week.	102	44	90	1,394	0	....
																			Third week..	130	46	84	1,440	0	0.11
																			Discharge.... (house diet).	163	65	88	1,724	0	0.11
Average		**61**	...	.....	**5.6**	...	.....	.....	.....	.....	.....	.....	...	..	..	...	...	.....	Preoperative	99	53	84	1,359	19	0.21
																			First week...	91	42	74	1,194	28	0.20
																			Second week.	103	51	86	1,389	23	0.17
																			Third week..	108	54	89	1,455	18	0.17
																			Discharge....	120	59	96	1,565	7	0.17

diabetes, which usually is made temporarily severe by the infection. In table 1 are summarized the records and diets of sixteen patients treated during 1929.

These sixteen cases illustrate the common problems in diabetic gangrene. Two patients under 50 years of age had had diabetes nine and fourteen years. Syphilis was present in case 7652. In six cases diabetes had been discovered only within the year, because no urinary tests had been made previously by a physician. Earlier diagnosis with instruction in the care of the feet might have prevented, or at least postponed, gangrene for years. Angina pectoris was present in three cases. All patients had albuminuria of slight degree. The nonprotein nitrogen of the blood was above 40 mg. per hundred cubic centimeters in three cases. The systolic blood pressure exceeded 150 in four cases and was below 120 in two cases.

TABLE 2.—*Average Daily Diets Used in Cases of Diabetic Gangrene Before and Since the Use of Insulin*

		Before Operation			After Operation											
					First Week			Second Week			Third Week			At Discharge		
No. of Cases	Date	Carbohydrate	Calories	Insulin	Carbohydrate	Calories	Insulin	Carbohydrate	Calories	Insulin	Carbohydrate	Calories	Insulin	Carbohydrate	Calories	Insulin
7	1921-1922	44	543	0	66	681	0	53	838	0	55	923	0	77	1,164	0
10	1925-1926	80	1,372	18	82	1,323	24	87	1,445	20	91	1,531	15	97	1,677	3
16	1929	99	1,359	19	91	1,194	28	103	1,389	23	108	1,455	18	120	1,585	7
Per cent increase in carbohydrate and calories, 1929 over 1921-1922.....		125	150		38	75		94	66		96	57		56	36	

In table 2 the average diets and dosages of insulin for the group are compared with those in similar cases in earlier periods. In 1929, the average amount of carbohydrate in the diets at discharge exceeded that in 1922 by 56 per cent, owing to the use of insulin in large measure. However, if one selects the seven patients who on discharge from the hospital in 1929 had discontinued the use of insulin (table 1), the average amount of carbohydrate in the diet on discharge was 140 Gm. as compared with 77 Gm. in the diet of similar patients discharged in 1922. One may reason that the difference was due to the inherent mildness of the diabetes or to a less severe infection in the later group. On the other hand, better management of the disease may have brought about improved resistance. In case 7884 the patient required 50 units of insulin daily before operation, yet when he was discharged his diet contained 89 Gm. of carbohydrate and insulin was not used. In case 7759 the patient required 75 units daily before operation, but when he left the hospital he was taking but 5 units. The capacity for

improvement even of patients with gangrene is far greater than was supposed. At present (August, 1930) most patients receive 150 Gm. of carbohydrate before discharge.

So striking are the changes in dietary treatment from one period to another that it is obvious that still greater improvement may occur. Some definite information regarding the best diets in cases of diabetic gangrene is desirable. Before operation, the existence of fever and the generally disturbed state of the patient require the use of the simplest food in amounts which the patient can retain. After operation, however, the problem of determining the optimal diet arises. The basal metabolism in cases in which toes or legs have been amputated as compared with the total calories of the diets is shown in table 3. In

TABLE 3.—*Postoperative Basal Metabolism of Eleven Diabetic Persons*

Case Number	Sex	Age	Activity at Time of Test	Date	Calories in Diet	Deviation from Standard, per Cent	Pulse	Computed Calories per 24 Hours	Weight, Pounds	Period Since Operation	Operation: Amputations	Operation: Other
5155	M	45	Normal	4/ 3/26	1,576	— 4	80	1,829	194¼	24 years	2 legs*	
4396	M	63	Bed	1/28/25	1,596	+10	67	1,441	135	33 days	1 toe	
4396	M	63	Chair	4/ 5/26	1,561	+ 7	88	1,320	114	23 days	1 leg	
4396	M	63	Walking	4/26/26	1,553	+25	74	1,549	114	46 days	1 leg	
5186	M	60	Bed	4/ 5/26	1,751	+12	88	1,390	116½	14 days	2 legs	
5163	M	59	Chair	4/ 5/26	1,568	— 2	76	1,223	118½	23 days	1 leg	
5250	M	60	Bed	4/ 8/26	1,748	+40	..	1,355	80½	47 days	2 legs	
6643	F	62	Chair	2/ 3/28	1,369	— 1	64	1,035	92	67 days	1 toe	
4420	M	65	Chair	2/ 4/28	1,927	+22	78	1,963	176¼	18 days	1 toe	
6088	M	68	Bed	2/ 8/28	1,587	+ 1	60	1,211	122½	30 days	.....	Carbuncle
6656	M	67	Bed	2/14/28	1,898	+19	92	1,575	135½	27 days	1 leg	
6648	F	61	Bed	2/15/28	1,505	+25	76	1,461	114½	21 days	1 toe	
6790	M	61	Bed	2/16/28	1,686	— 7	68	1,174	121¾	24 days	1 toe†	

* Amputations below the knee at the age of 21.
† Gritti-Stokes amputation on March 19, 1928.

case 5155 the patient was not convalescent; he was in the hospital only a few days, and a comparison of his diet and his computed metabolism is not proper. His data are included to show that the basal metabolism of a legless diabetic patient, uninfluenced by the effects of a recent infection or operation is about 21 calories per kilogram of body weight. Three determinations of the metabolism in case 4369 showed some variation. The first test was done before the patient's leg was amputated. The second test was done a year later, when he had lost some weight. With a total loss of 21 pounds (9.5 Kg.), the loss in metabolism was but 121 calories. In the third test the metabolism was so much higher as to seem aberrant, but there was no reason to doubt it. In case 5250 both legs were amputated through the thigh because of gangrene and severe infection. The patient was extremely emaciated, but was not nervous and did not have a severe case of diabetes. His high metabolism was probably due to the persistence of slight infection

in his wounds. The average deviation of the group from the Harris and Benedict standard predicted metabolism was + 12 per cent. The average twenty-four hour computed calories was 25 per kilogram of body weight. When one considers the lowering of the metabolism during sleep, which might compensate for the slightly increased metabolism associated with movements in bed, it is probable that most patients with gangrene when free from active infection will retain their weight if given sufficient food to supply 25 calories per kilogram of body weight. For certain emaciated or debilitated patients, additional food might be necessary. The caloric needs of persons must be estimated not only from computations but after some consideration of their physical make-up, appetite and indications shown by their progress from day to day. The composition of the diet will also depend on complications such as nephritis and also on variations in the caloric tolerance of the

TABLE 4.—*Diet Before Operation*

Food	Breakfast	Dinner	Supper	Total, Gm.	Carbohydrate	Protein.	Fat
5% vegetable	...	150	150	300	10	5	..
10% vegetable	...	75	75	150	10	3	..
Orange	...	150	150	300	30	..	..
Oatmeal	30	...	...	30	20	5	2
Uneeda	2	2	2	6	30	3	3
Potato	...	60	...	60	12	2	..
Egg	1	...	...	1	..	6	6
Cream, 20%	60	30	30	120	4	4	24
Butter	10	15	10	35	..	..	29
Meat	...	60	...	60	..	32	20
Grapefruit	200	...	200	10	..	..	..
Grams					126	60	84
Calories					4	4	9
Total calories, 1,500					504	240	756

patients. Rapid changes in the amount of carbohydrate in the diet may be made as tolerance improves, as shown in table 1, and more surely if the intake of fat is restricted.

Preoperative Treatment.—A liver well stored with glycogen is the diabetic person's best protection during an operation. He must therefore receive sufficient carbohydrate, but under such conditions that he will surely utilize it. The diet to be used before operation depends on the length of time available for preparation. In an emergency operation to be performed within a few hours of the patient's admission to the hospital, because of threatening sepsis, no attempt should be made to force foods. It is worth while, however, to have the patient receive about 20 Gm. of carbohydrate in the form of 200 Gm. of orange juice or oatmeal gruel, made from 30 Gm. dry weight, with insulin. When a period of days elapses between the admission and the operation, the patients should receive a diet of approximately 100 to 150 Gm. of carbohydrate with 1 Gm. of protein per kilogram of

body weight and sufficient fat to give 25 calories per kilogram of body weight. Such a diet for a man weighing 132 pounds (59.9 Kg.) is shown in table 4.

If the urine contains sugar, and especially if diacetic acid is present, insulin should be given as soon as the patient is admitted to the hospital or as soon as the diagnosis of diabetes is established. If sugar and diacetic acid are present, it is usually proper to start with 10 units three times a day, provided sugar is present in the urine before every meal. If the urine becomes free from sugar, the insulin should be reduced to 5 units before the next meal; if the next specimen of urine is free from sugar, the dose should be omitted. At the time of operation it is rarely necessary to give insulin during the actual operation; two or three hours before, my associates and I usually give 15 Gm. of carbohydrate in the form of 150 cc. of orange juice and 10 or 15 units of insulin, unless the patient is accustomed to much larger doses. Even then, it has not been necessary to use more than 20 units before operation.

Postoperative Treatment.—Vomiting rarely occurs after operation. If spinal anesthesia has been used, the patient can begin taking food immediately. In cases in which general anesthesia is followed by vomiting, hypodermoclysis of 1,000 cc. of 5 per cent dextrose solution in physiologic solution of sodium chloride or an intravenous injection of 500 cc. of 10 per cent dextrose solution in physiologic solution of sodium chloride may be used to replace fluids, give nourishment and check the nausea.

The usual diet is simplified by the use of more concentrated foods, such as cereal, potato, rice and toast, in such quantities that the total amount of carbohydrate in the diet is unchanged. The urine is tested every four hours, and the insulin is given according to the amount of sugar present. If the urinary test is red or orange with Benedict's solution, then 15 units of insulin may be given; if it is yellow, 10 units; if green, 5 units. The order for insulin is written contingent on urinary tests, so that unnecessarily large doses of insulin will not be given as a routine after the patient becomes free from sugar during the two or three weeks following amputation. If the urine becomes sugar-free, additions of 10 Gm. or more of carbohydrate to the prescribed diet may be made from day to day to keep pace with the growing carbohydrate tolerance. Gains in tolerance depend on the removal of infection, the constancy and regularity of the diet, the use of insulin and perseverance in seeking to increase the portion of carbohydrate in the diet. The use of exercises in bed improves both muscles and morale. They are begun within a few days after the operation, provided the patient is free from a high temperature. At first they consist only of breathing exercises and of motions of the arms. Gradually the number of exercises are increased, and the remaining leg shares the

exercises. Hand elastic exercisers are attached to the foot of the bed, so that the patient may exercise more vigorously as he gains in strength.

Use of Dextrose Solutions.—For patients in a state of shock or so weakened that they cannot take food by mouth, the use of dextrose given by rectum, subcutaneously or intravenously, is of great value. Rectal injections can be used but a few times because of the irritability of the rectum. Dextrose given intravenously is rarely needed when spinal anesthesia has been used. However, in some cases it is of great value as a means of getting nutrient substances into the body and as a stimulant to the circulation. Ordinarily, it is given in 10 per cent solution in amounts of from 500 to 1,000 cc. once or twice a day. Precautions must be observed in its use owing to the fact that, when insulin is given with dextrose and little other food is being taken, hydroglycemia may follow a few hours after injections. Hypoglycemia seems to be caused by a stimulation of the pancreas by the dextrose. When a patient is being fed exclusively by intravenous injections of dextrose solution, orders for the administration of insulin should be so written that insulin cannot be given unless a test of the urine is made, and unless the dose is adjusted according to the presence or absence of sugar in the urine.

Laboratory Aids.—A blood culture should always be taken when the patient has gangrene and fever. If septicemia is suspected, the absence of sugar in the urine is usually strong evidence against its existence, and this may be a decisive point in deciding the type and the time of operation. The absence of sugar in the urine must never be taken as proof of the absence of diabetes, since the dextrose threshold in the blood is often elevated in persons with diabetes of long duration and in older diabetic persons. If hyperglycemia is found, the surgeon is thereby warned that vascular disease in the extremities is probably of the diabetic rather than of the purely senile type, and that diabetic treatment may be valuable and even life-saving. Such patients may develop dangerous acidosis and even coma after operation if precautions are not taken. Diacetic acid should always be tested for by means of the ferric chloride reagent whenever the urine is examined for sugar. Only in this way can the surgeon or the physician recognize the development of acidosis in its earliest stages.

Blood Sugar.—Variations in the blood sugar are the most valuable single indication of variation in the diabetic metabolism. When the amount of blood sugar is between 0.1 and 0.15 per cent, the patient is for the time secure from acidosis and hypoglycemia. The higher the amount of blood sugar before operation, the more serious is the general condition of the patient and the worse the prognosis (table 5). There is no level of blood sugar, however, which necessarily makes the case hopeless. The highest amount of blood sugar is found in severe aci-

dosis, and a figure above 0.4 per cent in a surgical case should always suggest approaching coma, whether before or after operation. In many cases of long duration, with advanced arteriosclerosis and acidosis, the lower the amount of sugar in the urine the higher the amount of sugar in the blood. Anesthesia produces elevation in the blood sugar, the greatest change being caused by ether, the next greatest by nitrous oxide and the least by ethylene. Insulin reduces the sugar in the blood at a rate which varies somewhat with the severity of the diabetes, the presence of infections, the size of the dose of insulin and the previous diet of the patient. The maximum lowering of the blood sugar after a dose of insulin is found from three to six hours after the injection. It is almost impossible to predict with certainty the amount of lowering of the blood sugar that a given dose of insulin will cause. In diabetic persons

TABLE 5.—*Duration of Life of Patients Surviving Amputations of Toes or Legs Between Jan. 1, 1923 and Jan. 1, 1928 Compared with the Percentage of Sugar in the Blood*

Blood Sugar, per Cent	Patients	Gangrene: Living, August, 1929		Average Duration of Life, Dead	
		Number	Years	Number	Years
Gangrene					
0.30 to 0.45	14	6	2.2	8	1.2
0.20 to 0.29	44	22	2.6	22	1.2
0.10 to 0.19	20	8	3.2	12	1.1
Infection					
0.30 to 0.45	9	3	2.0	6	0.8
0.20 to 0.29	18	14	2.1	4	1.9
0.10 to 0.19	19	8	3.8	11	1.1

changes occur from hour to hour. The diet and insulin must be adjusted daily, even hourly, in emergencies, according to the urinalyses or blood tests.

Reactions to Insulin.—Hypoglycemic reactions are serious, and are to be avoided at all costs. They endanger the patient's life, because they threaten his diseased heart. The symptoms of hypoglycemia are weakness, severe hunger, tremor, convulsions or unconsciousness usually occurring from three to eight hours after a dose of insulin. Queer mental states or absolute unconsciousness may develop with slight or no warning. The prevention of hypoglycemic reactions depends on constant watching of the urine, the diet and the blood sugar, especially in patients having less than their usual diet or receiving dextrose solution intravenously. It is wise to test the blood sugar at those times in the day when hypoglycemia is most likely to develop, namely, at 11:30 a. m., 4:30 p. m. or 9 p. m. If the urine of a patient given insulin three times a day is tested every six hours and is found to be sugar-free once in the day, that is the time when hypoglycemia is most likely to occur. If the blood sugar level is found to be below 0.1 per cent at this time, it is well to reduce the dose of insulin from that

given four hours before, even though no symptoms have been recognized, or else to have the patient take 10 Gm. of carbohydrate in the form of orange juice or two Uneeda crackers. Before the patient's discharge from the hospital, the blood sugar test should be done at 11:30 a. m. or 4:30 p. m. in order to avoid the occurrence of hypoglycemia at home.

Treatment for hypoglycemia should be instant. In mild cases, 100 cc. of orange juice may be given by mouth. When this fails or the patient is unconscious, dextrose solution (20 cc. of a 50 per cent solution) should be injected intravenously. The hypodermic injection of 0.5 cc. of epinephrine (1 : 1,000) sometimes brings the patient to, so that he can take orange juice or Karo syrup.

Acidosis.—In every case of gangrene coma is likely to occur. If the patient vomits, becomes drowsy or has pain in the abdomen, coma should be considered first, and the urine should be tested for sugar and diacetic acid. When diacetic acid is present in the urine, a carbon dioxide-combining power in the blood plasma of from 35 to 50 per cent by volume means mild acidosis; when the percent is below 20, the patient is usually in obvious coma. The onset of coma is insidious, with loss of appetite, nausea and pain in the abdomen. Air hunger is a late symptom. Treatment with insulin should begin long before air hunger develops, and should be as promptly and energetically carried out as would be treatment for intra-abdominal hemorrhage. Gastric lavage, an enema and subpectoral infusion of saline are essential. From 10 to 25 units of insulin hourly or even half hourly is usually necessary until glycosuria is controlled.

Complications.—Arteriosclerotic disease of the heart and kidneys far exceeds all other complications. Most important is the protection of the skin against boils and carbuncles and of the feet against pressure sores. Scrupulous cleanliness of the skin, use of nonirritating soap and frequent changes of position to avoid the macerating effect of pressure and perspiration help to prevent carbuncles in obese patients. When small furuncles appear, ultraviolet treatment is valuable. All patients who have had a leg amputated should have a thick clean woolen sock on the other foot to avoid pressure of the heel against the bed. A Balkan frame over the bed with hand grips hung from the frame makes it possible for a patient to use his arms to turn instead of digging his heel into the bed for this purpose.

PROGNOSIS

The prognosis for patients with lesions of the feet depend on: (1) the nature and severity of the infection, (2) the condition of the blood supply of the foot and (3) the opportunity for adequate treat-

ment for the diabetes and complications. All lesions of the lower extremities in the diabetic patient are classified etiologically as chiefly due either to infection or to a deficient blood supply. Patients whose condition is primarily due to infection usually have warm feet and palpable dorsalis pedis pulse; patients with deficient circulation usually have no pulsation in the dorsalis pedis artery and cold feet. A more complete discussion of this classification is given in an article by Dr. L. S. McKittrick. The two groups differ in many respects. The average age of patients in the infectious group was 58.8 years, and the duration of the diabetes 8.5 years. The average age of those in the circulatory group was 63.5 years, and the duration of the diabetes 7.5 years. The prognosis in the two groups differs widely, and in discussing various factors concerned with prognosis in diabetic gangrene the cases should be grouped under these two headings.

The thirty-one deaths occurring in the hospital illustrate the four most unfavorable prognostic criteria: (1) virulent infection in feet with absent dorsalis pulsation and a deficient blood supply (twenty-five belonged to the circulatory group); (2) advanced age, the average being 69 years; (3) long duration of diabetes, the average being 8.9 years; (4) angina pectoris and chronic nephritis. Four deaths from coronary thrombosis in cases of the gangrene group are more impressive when included with six deaths in this group occurring within a year after operation. A history of angina pectoris is easily missed. The pressure or pain over the sternum may be slight, but if it bears a definite relationship to exertion or nervous excitement, the diagnosis cannot be doubted. Electrocardiograms often give definite evidence of marked myocardial change, presumably secondary to coronary disease, even when attacks of angina have not occurred. Sudden death is an imminent possibility in any diabetic person after 60 years of age.

In contrast to the patients who died in the hospital are thirteen patients with gangrene living an average of 4.2 years after operation. Their average age was 59 years as compared with the average of 63.5 years for all cases of gangrene. The average duration of the diabetes was 5.2 years in contrast to an average duration of 8.5 years for the entire group. The average amount of blood sugar was 0.2 per cent and the average amount of sugar in the urine on admission was 0.4 per cent. In four cases the systolic blood pressure was above 150, and on only one occasion was it under 110. In no case was the nonprotein nitrogen above normal, and in no case was there suggestive evidence of severe coronary disease. The infectious group, in which the average age was 60.2 years and the average length of life 4.3 years after operation, consisted of five males and two females. The average duration of diabetes was 5.9 years as contrasted with a duration of 7.5 years in the entire group of infectious cases. In four cases of this group

amputation of the toe was performed and in three amputation of the leg. Again, the average amount of blood sugar was 0.2 per cent and the average amount of sugar in the urine 1.1 per cent. In two instances, the nonprotein nitrogen was above 40 mg. per hundred cubic centimeters. In no case was angina pectoris present. Younger patients with diabetes of shorter duration, the absence of septicemia and advanced coronary disease are the reasons for a better prognosis.

The prognostic value of the purely diabetic factors is paradoxic. The more severe the disturbance, the greater the relief that will be afforded by adequate and successful treatment. The level of the blood sugar and the amount of sugar in the urine on admission to the hospital are not closely related to the ultimate prognosis, owing to the fact that they may be influenced so markedly by treatment during the week or two preceding admission. Much more important than the level of blood sugar at the time of admission are the course and treatment of the diabetes during the preceding five or ten years. It is true, however, that an excessively high blood sugar has a somewhat unfavorable significance, as seen from table 5.

In general, the level of the blood sugar is so much influenced by infection and the presence or absence of recent treatment that its chief value is as a guide to diabetic treatment.

The blood pressure is of no outstanding importance in relation to prognosis. In 50 per cent the systolic blood pressure exceeded 150 mm. Hypotension with a systolic pressure below 110 occurred in only six cases. So far as a prexisting hypertension may have paved the way for coronary disease or chronic nephritis it is of importance, but at the time of treatment in the hospital the level of the blood pressure seemed to have little bearing on the outcome. Actual cardiac failure with decompensation has occurred in only two cases in the hospital.

The only two cases in which gangrene developed, in spite of fairly good care of the feet, were cases associated with angina pectoris and chronic myocarditis.

EXPLOSIONS OCCURRING DURING THE USE OF ETHYLENE*

FRANKLIN D. JOHNSTON, B.S. (ENG.), M.D.
ANN ARBOR, MICH.
AND
HUGH CABOT, M.D.
ROCHESTER, MINN.

A few months after Luckhardt and Carter had published their articles on the original clinical work with ethylene, Brown[1] reported the results of an extensive investigation concerning the explosibility of ethylene mixtures. In this paper, he mentioned the places where explosion may occur as:

(1) While the gas is compressed in the cylinder; (2) after emerging from the cylinder but not mixed with oxygen; (3) in the mixing chamber or distributing apparatus mixed with oxygen and (4) after escaping into the room through the valve in the facepiece.

In the first two locations one is presumably dealing with the pure gas, and whether or not explosions can occur at these points depends on the inherent character of the gas. In order to settle this question definitely, one must know whether it is possible to cause the pure gas to explode or whether it can explode spontaneously. Some gases, such as acetylene, are unstable when highly compressed. Does ethylene show such properties? Experience has shown that ethylene is not unstable when under high pressure, and that the pure gas cannot be made to explode. Brown[1] has shown that ethylene unmixed with oxygen will not explode, and, furthermore, that explosions do not occur in mixtures of ethylene and oxygen unless at least 40 per cent of the mixture is oxygen. This work was confirmed by Hornor and Gardenier,[2] although their results show that ethylene oxygen mixtures containing a minimum of 30 per cent of oxygen may explode. The criterion, however, established by these workers was rigid. If the mixture was not exploded at once by a spark, a continuous arc was passed through it for one minute before it was definitely said to be nonexplosive.

---

* Submitted for publication, May 2, 1930.

* From the University Hospital, Ann Arbor, Mich.

1. Brown, W. E.: Explosibility of Ethylene Mixtures, J. A. M. A. **82**:1039 (March 29) 1924.

2. Hornor, A. P., and Gardenier, C. B.: Anesth. & Analg. **7**:69 (March) 1928.

Additional evidence showing that pure ethylene or very concentrated mixtures of the gas are not explosive is found in the observation of Herb [3] that following an explosion in the distributing portions of the anesthetic apparatus, there is no tendency for the explosion to flare back into the machine or up to the tanks. On several occasions the pure gas coming from the tank was ignited, but the flame could be easily extinguished. Moreover, we must remember that although the use of ethylene by the medical profession is recent the gas has been known to chemists and used in industry for many years. If the pure gas were unstable or if its use were fraught with danger of mishap, warnings of these dangerous properties would have been forthcoming. The fact that many thousand drums of the highly compressed gas have been shipped to all parts of the country without accident argues well for its stability.

Recently a terrible accident occurred in an Indiana hospital.[4] A physician working with a gas machine carrying tanks of oxygen, nitrous oxide and ethylene was killed when a violent explosion occurred. The accident was reported in the papers as due to an ethylene explosion. Subsequent investigation proved that the tank of nitrous oxide and not the ethylene tank had blown up. That matter is of secondary importance, however, to the fact that the tragedy proved the extreme instability of mixtures of nitrous oxide and ethylene under pressure. In gas machines of the older type, when the valves on the tanks are not tightly closed, contamination of certain tanks with gases from other tanks on the same machine may occur. The fact that mixtures of nitrous oxide and ethylene in certain proportions may be extremely explosive has not been sufficiently emphasized. Circulars informing the medical profession of this danger and also mentioning the possibility of the gases mixing in the tanks when attached to certain old style machines were sent out by a chemical supply house to users of their products. Hewer, in two publications,[5] mentioned the fact that ethylene forms explosive mixtures with both oxygen and nitrous oxide. Hornor and Gardenier [2] showed that mixtures of nitrous oxide and ethylene are explosive when the latter gas is present in concentrations of from 5 to 35 per cent. Above concentrations of 35 per cent ethylene, explosions do not occur. Thus we see that small amounts of nitrous oxide in ethylene will not lead to explosion but that small amounts of ethylene in a nitrous oxide tank

3. Herb, I. C.: Explosions of Anesthetic Gases, J. A. M. A. **85**:1788 (Dec. 5) 1925.

4. Personal communication to Dr. Cabot.

5. Hewer, C. L.: Lancet **1**:173 (Jan. 24) 1925); Brit. J. Anæsth. **3**:174 (April) 1926.

may be very dangerous. On this basis alone, we can understand why it was that a nitrous oxide tank and not an ethylene tank blew up in the recent accident in Indiana.

In view of all the evidence that has accumulated on the subject it seems that pure ethylene will not explode either when ignited as the free gas or spontaneously when compressed in steel cylinders.

Since it is clear that the pure gas does not explode, all the operating room accidents that have occurred with ethylene must have been due to the ignition of mixtures of ethylene and oxygen in the mixing chamber of the machine, in some distal portion of the apparatus, or out in the room after the gas has been exhaled by the patient. Before considering explosions occurring in these different places, we must ask ourselves an important question.

Can mixtures of ethylene and oxygen explode spontaneously? Chemical mixtures are known that are so exceedingly unstable that an explosion may occur with no apparent cause. Other mixtures may explode if some slight chemical or mechanical stimulus is provided. Sise [6] stated that mixtures of ethylene and oxygen under high pressure, as would be true if ethylene should leak into an oxygen tank, may explode spontaneously. The recent disaster in Indiana proved the same thing to be true of mixtures of ethylene and nitrous oxide. However, it is fairly evident that at normal pressures even the most explosive concentration of ethylene in oxygen will not blow up in the absence of a definite exciting cause. A great deal of experimental work has been done, employing low concentrations of ethylene in oxygen without accident, and during practically every ethylene anesthesia, especially toward the end when the patient is flooded with oxygen, the concentration of the gases must at some time be at the optimum point for explosion to occur. Moreover, the gases are not stationary but are passing fairly rapidly through a closed system, being thereby subjected to mild traumatic influences. The most satisfying argument against the theory of spontaneous explosion, however, is found in the fact that for all the explosions that have occurred a tangible exciting cause can be found. The cause has been either actual flame or cautery near the anesthetic apparatus or in many cases a static spark occurring either inside the machine and distributing hoses or out in the operating room. Explosions occurring outside of the machine will be considered first.

EXPLOSIONS OCCURRING IN THE OPERATING ROOM

Brown,[1] in his early report on the explosibility of ethylene mixtures, concluded that explosion due to gas that had escaped into the

6. Sise, L. F.: Boston M. & S. J. **192**:287 (Feb. 12) 1928.

room was a negligible possibility. He based his view on a theoretical calculation which showed that in the average-sized operating room and with average pulmonary ventilation by the patient six hours would be required before the concentration of ethylene in the room could reach the explosive level of about 5 per cent. He assumed no ventilation in the room, a factor usually always present to a variable extent. He also assumed uniform diffusion throughout the room, while in reality, since the gas is but slightly lighter than air, it probably diffuses slowly and forms currents or strata much like those formed by tobacco smoke in a still room. Under these conditions it is possible to understand how an explosion could occur. Davis [7] and Caine [8] reported explosions occurring in the room some distance from the patient and the gas machine, and Sise [6] commented on the possibility that exists for such accidents to occur.

It is obvious that the mere presence of even highly explosive mixtures of ethylene and oxygen in the operating room will do no harm, provided no agent is present that may ignite the gases. The explosions that have occurred during the use of the cautery might well have been caused by careless use of roentgen equipment, a spark produced on the brushes of an electric motor or a spark produced when an electric circuit of any sort is opened. These are perfectly evident sources of danger not only with ethylene but with ether as well. Sparks caused by electrostatic charges may be equally dangerous. They are, as a matter of fact, a much greater hazard than the obvious things; not because every static spark means an explosion but because they belong to a world unseen and may be generated in so many different ways. A static spark always results and can be produced only when two bodies of different electrostatic charges are separated or brought together. The charge must always be present before the spark and, unfortunately, nature has not given man the sense organ that can perceive when objects about us are charged. To combat such a situation we must adopt indirect methods and fall back on our knowledge of the fundamentals of electrostatics. A consideration of this matter in detail will be found later in this paper.

To eliminate the danger from explosions occurring out in the operating room, two possibilities must be considered. The explosive gases can be prevented from getting out in the room where they might be ignited, or measures can be taken to remove from the operating room any possible source of ignition. The latter method is the one

7. Davis, C. H.: Explosibility of Ethylene, J. A. M. A. **82**:1607 (May 17) 1924.

8. Caine, A. M.: Anesth. & Analg. **3**:104 (June) 1924.

usually adopted. Use of the cautery or any apparatus through the action of which a spark may be produced is strictly prohibited in the operating room where ethylene is employed. The weak point usually found in this method is that precautions against static spark are not taken and sooner or later an explosion occurs. Ethylene is blamed for the trouble (which might have occurred with ether vapor as well), and its use is curtailed. Means for eliminating the hazard of static spark in the operating room will be considered later.

The other method of preventing explosions in the operating room is to prevent ethylene from entering the room either by the use of a hose connecting the exhaust valve of the facepiece to the outside or by the use of a closed system. Salzer [9] described a system of the first type. He uses an attachment which fits over the expiratory valve of the McKesson facepiece. To this device is connected a noncollapsible tube leading to the outside. The arrangement is simple and easily installed, but to be sure that it accomplishes its purpose, leakage of gases around the cushion on the patient's face would have to be prevented.

The system of rebreathing described by Gatch [10] is used exclusively by some anesthetists, and if the rebreathing is complete, the amount of gas escaping into the room is said to be insignificant. Poe [11] used such a system in a series of 8,884 ethylene cases with great satisfaction and without mishap. He employs the Sander's modification of the Gatch apparatus, which has been used in approximately 185,000 administrations of ethylene in Texas and other states without an explosion occurring.

A strictly closed system in which the respiratory carbon dioxide is absorbed in soda lime, and oxygen and the anesthetic gas are admitted in small quantities as needed provides a means whereby no ethylene (or other gas) can get out into the operating room. This type of equipment has been used on the continent for the administration of acetylene, and it possesses the disadvantages of being both cumbersome and expensive. It should be pointed out, however, that the closed systems and, to a lesser extent, administration with rebreathing allow the use of considerably smaller quantities of the anesthetic agent. In the closed system the same ethylene could be used over and over.

---

9. Salzer, M.: Method of Eliminating Danger of Explosion in Use of Ethylene, J. A. M. A. **88**:315 (Jan. 29) 1927.

10. Gatch, W. D.: The Use of Rebreathing in the Administration of Anesthetics, J. A. M. A. **57**:1593 (Nov. 11) 1911.

11. Poe, J. G.: Anesth. & Analg. **7**:295 (Sept.-Oct.) 1928; Text on Anesthesia, New Orleans, J. A. Majors Company, 1926.

We must mention here the fact that the means outlined to prevent explosions from free ethylene in the operating room apply also to gas-oxygen mixtures with ether vapor. Poe[12] stated that mixtures of ether and oxygen are highly explosive, much more so than ether in air. Brown,[1] and Hornor and Gardenier[2] gave figures showing the wide range of ether-oxygen mixtures with and without nitrous oxide which will explode. It is true that ether vapor, being heavier than air, sinks to the floor and to some extent out of harm's way, but that some explosions attributed to ethylene must in reality be blamed on ether cannot be doubted. Guthrie[13] mentioned "a most unfortunate and widely heralded fatal explosion" which actually occurred while nitrous oxide and ether vapor were being given, although ethylene had been used earlier in the anesthesia.

Our discussion of ethylene explosions up to the present has been limited to those which occur not in the anesthetic apparatus but out in the operating room. Except for the matter of static charges acting as the causative agent in these explosions, it is evident that such accidents are readily preventable by ordinary common sense and a few precautions. Explosions within the machine or accessory parts can be due only to ignition by something inside the apparatus. Static spark is the only agent that can be seriously considered in this connection. Because static charges are responsible for many accidents occurring in the operating room and must be entirely blamed for the explosions occurring within the machine, it is plain that time expended in a careful study of electrostatics and the electrical characteristics of the anesthetic equipment is time well spent.

ELECTROSTATICS AND THE ELECTRON THEORY

It is essential to know at the outset the modern conceptions of the nature of static electricity, how it may be generated and why sparks occur. The electron theory is a simple and adequate means of explaining these phenomena.

According to the electron theory, all matter is made up of atoms, each atom containing a positively charged nucleus about which are grouped in different configurations a varying number of electrons. The arrangement and number of electrons grouped about a nucelus depends on the type of atom with which we are dealing. Thus elements of low atomic weight are made up of atoms having relatively few electrons of simple space arrangement, while for elements of high atomic weight the reverse is true. The electron is conceived to be the smallest

12. Poe (footnote 11, first reference).
13. Guthrie, D.: Surg. Gynec. Obst. **43**:703 (Nov.) 1926.

unit of negative electricity, and since ordinary matter is without electric charge, there must exist in every atom of such matter just enough electrons to neutralize the positively charged nucleus. Suppose that by some means one can rub off or displace from the atoms composing a substance some of the electrons that had hitherto been bound in the atoms. Obviously, if this can occur the positively charged nuclei of the atoms will no longer be neutralized, and the matter will possess what is called a positive charge. Thus it is seen that what is called a positive charge on a body is simply a deficit of electrons or negative electricity. In the same way, a negatively charged body possesses more electrons than are normally attached to it. The familiar experiment of charging an ebonite rod by rubbing it with fur provides an excellent example of this theory. When the ebonite rod is rubbed vigorously with the fur, presumably the friction (energy is required) causes some of the electrons in the atoms of the fur to be dislodged and transferred as free electrons to the ebonite rod. The rod therefore holds an excess of electrons and is negatively charged, while the fur atoms are lacking in some of their electrons and the fur itself is positively charged. From the mechanism involved, it is evident that the amount of negative charge on the ebonite rod must be exactly opposite and equal to the positive charge on the fur. If the charged ebonite and fur are placed in intimate contact for a time, both lose their charges since the free electrons on the ebonite return to their proper places in the fur atoms.

Experience has shown that charges are obtained on rubbing two bodies together only when (1) the substances are of different material, (2) they belong to the class of substances called insulators, and (3) they are dry. Concerning the first prerequisite we can say nothing, except that it is true. Glass cannot be charged by rubbing it with glass, nor does friction of rubber on rubber lead to charge. The second statement needs some explanation.

If one should rub together a bar of iron and a bar of copper held in the two hands, it is possible that charges may be produced on the two metals, but no charge could be demonstrated because these substances conduct electricity, which means that the electrons can move through them without much difficulty. If this is true, any excess of electrons produced by friction on one of the rods can flow around to their original positions through the body of the experimenter. Suppose, however, one rubs a piece of rubber against a steel rod. Friction of the metal on the rubber causes a displacement of free electrons from the steel onto the rubber, so that the latter is negatively charged and the former must possess an equal and opposite positive charge. But

here is a point of great importance: The negative charge on the rubber can be easily demonstrated at the place where the friction occurred but no charge is found on the metal. The answer is very simple, of course, but of great importance in understanding phenomena of exactly the same nature which occur on anesthetic machines. The charge is found on the rubber because an excess of electrons was rubbed off onto it and then these extra electrons could not get away. They stay on the rubber at the point where the transfer occurred because rubber is a nonconductor. It offers great resistance to the passage of electrons through it. No positive charge can be found on the metal rod, because it is a conductor and in direct contact with the body of the observer, which is also a conductor. Assuming the observer to be insulated from the ground, the positive charge will spread uniformly over the entire surface of the observer and rod so the charge at any point is so small as to be impossible of detection.

The reason why insulating substances must be dry in order that static charges may be produced should be evident from the foregoing paragraph. Even a thin film of moisture on the surface of an insulator gives it the characteristics of a conducting body as far as static charges are concerned. Thus static charges by their very nature can exist (be static) only when an insulating barrier is provided. If one touches a highly charged but insulated metal ball with a copper wire leading to the earth the charge on the ball is instantly dissipated. There occurs a transient flow of electrons up or down the wire, as may be necessary for the charged ball to be rendered electrically neutral. This flow of electrons is an electric current.

Static charges, of course, may be generated on an insulated body, or on only a small part of it, by friction with an unlike substance. The size of the charge varies with the amount of energy expended in rubbing the two bodies together, and whether or not the charge remains localized at the site where the friction occurs depends on the insulating properties of the substances involved.

When a body possesses an electrostatic charge, either positive or negative, space surrounding the object is under a state of strain which is called an electrostatic field. This is proved to be true by many phenomena that occur in space near charged objects. Thus mechanical forces of attraction or repulsion are exerted between charged bodies; conductors brought near a highly electrified object become charged themselves. The latter phenomenon is called charge by induction, and it may be of great importance in the present problem. Charge by induction is represented in figure 1.

A metal ball, *A*, is assumed to be strongly charged by a great excess of electrons; i. e., it is negatively charged. This charge cannot be lost because the ball is insulated from its surroundings by the glass standard on which it rests. An elongated metal shell, *B*, also supported on a glass rod, is now brought close to *A*. Investigation of the shell with an electroscope shows that its two extremities are highly charged, the side toward *A* possessing a positive, and the opposite end an equal negative charge. If the shell *B* is now withdrawn out of the field produced by *A*, it will be found to possess no charge. In other words, simply by placing this conductor near a charged body, one has produced a transient displacement of the electrons in its substance, a change that immediately disappears when it is drawn out of the electrostatic field. It should be evident that if the shell *B* were made of glass or hard rubber instead of metal, no such changes could occur because in these insulating substances the electrons are not free to move.

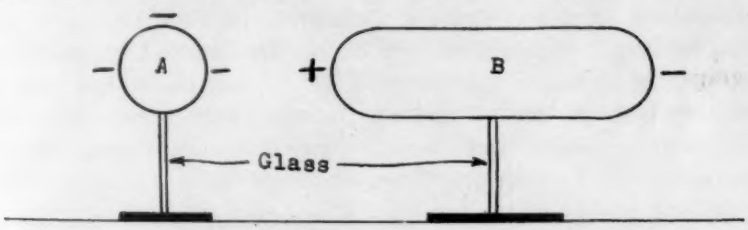


Fig. 1.—Sketch illustrating electrostatic charge by induction.

Suppose now that one continues to bring the metal shell *B* and the charged ball together until only a very short gap lies between them. Charges will be induced on the two ends of *B*, as we have described, and if one possessed means for measuring it, one would find an appreciable force of attraction between *A* and *B*. If one shortens the gap still more and watches closely, a spark will be seen to jump between the two conductors. A static spark has occurred. As the charges are approximated, the electric field between them becomes more intense until finally the insulating power of the air breaks down and a stream of electrons passes over the gap, leading to a more or less perfect equalization of the charge over both conductors. Investigation of *B* after the spark has occurred will show that it now possesses a negative charge spread over its surface.

In the foregoing paragraph we have assumed both the charged ball and the shell to be conductors of electricity. What will occur if one or both of these bodies are insulators? First, let it be assumed that the ball *A* is made of hard rubber and is highly charged as before with a negative charge. As the shell, which is of metal, approaches the ball,

charges will be induced on its extremities as described. Finally, as the interval between the two becomes very short, a spark occurs; but this spark is very small and has little energy behind it. The reason is found in the fact that although the rubber ball possesses on its surface a tremendous excess of electrons which it would like to get rid of, it cannot do this because they are bound by the insulating character of the ball to stay where they are. In other words, the excess charge has only been removed from the ball at the point of nearest approximation with the shell; to equalize the charges, as was accomplished by a single powerful spark from the metal ball, one would have to approximate, or even touch, the shell to the ball many times over a greater part of its surface.

If the charged ball were made of metal and the elongated shell of rubber, no induced charges would be noted as the two are brought together, and should a spark occur between them it would be very weak indeed. Again to equalize the charges on these two bodies one would be forced to touch, or very closely approximate, a great many points on the shell with the charged ball. If both objects are made of insulating material, the charges on the two could be equalized with even greater difficulty, and sparks of appreciable size would be hard to obtain.

All this discussion simmers down to something that all have experienced. As a child, one was accustomed to rub one's feet over the carpet during the cold winter months and then discharge oneself by touching the back of father's neck or some other convenient conductor of electricity. It was soon found out that the biggest spark was obtained by touching a radiator, the best and largest conductor in the room, and that no spark would result if one touched the wall or other insulating material. It was also found that no sparks were produced when persons touched each other, a phenomenon that demonstrates an all-important truth. It is not the magnitude of the charge on two objects that will determine whether or not a spark will occur between them, but it is the difference in electrical potential that is important. Sparks cannot occur between various metal parts on a gas machine if they are connected together electrically, no matter how highly charged the entire mechanism may be.

It has been shown that electrostatic induction plays an important part in the production of static sparks. It is further important in that pieces of metal, simply by being placed in the vicinity of a charged body, become charged themselves by induction and thus become a potential source of static spark. It is conceivable that a highly charged object merely by being adjacent to a gas machine could induce on the

latter charges that might lead to a spark and the ignition of explosive gases.

A static spark represents the release of a certain amount of energy that has been stored in the field surrounding a charged body. One sees the spark, one hears it, and, furthermore, energy is liberated in the form of heat which may suffice to ignite anything inflammable nearby. It is important to keep in mind that the static spark is only a means of equalizing differences in electrical potential. It is not unlike the rush of steam from the safety valve on a boiler. When the pressure difference, either steam or electric, exceeds a certain amount, the connecting medium breaks down and a rush of steam or electricity results. The ability of the spark to ignite an explosive mixture will depend on two factors: (1) The amount of energy dissipated in the spark and (2) the readiness with which the mixture explodes. The energy liberated by the spark will be greatest when the charge is very large and when the bodies between which the spark is to occur are conductors, so that all the energy stored up in the electrostatic field can be expended at once. We have shown that the explosibility of mixtures of ethylene and oxygen varies with the concentrations of the two gases present.

Let us now endeavor to apply to the present problem some of the principles that we have just laid down and see if we are not able by such a method to come to some conclusions concerning the rôle that static charges may play in the operating room. In the discussion that follows, much will be said that applies specifically to the McKesson Model G apparatus, and for that reason certain of our conclusions may not be true for machines of different make. The general principles will remain true for machines of all types, but structural variations make a great deal of difference in the theoretical possibilities.

Before entering on the next discussion, a word should be said about the well known fact that static spark occurs particularly in the winter months and, it might be added, in the colder parts of the country. The reason for these facts is, of course, found in the lower humidity in the winter months and in the colder climates.

APPLICATION OF THE ELECTROSTATIC THEORY TO THE ANESTHETIC MACHINE

The McKesson Model G machine is essentially a rugged and underslung platform made of steel and supported by three rubber tired wheels. The single wheel at the front of the machine is fastened at the lower end of the metal post on the top of which are located the mixing chamber, automatic shut-off valves and the mechanism that

supports the supply bags, one on each side of the metal post. To the front side of the mixing chamber, the ether chamber and the rebreather, all in one unit, are securely held by a metal coupling. At the opposite extremity of this device is the metal connection over which the hose leading to the patient is slipped. The gas tanks rest, of course, on the low platform and are supported by a chain attached to a stout metal

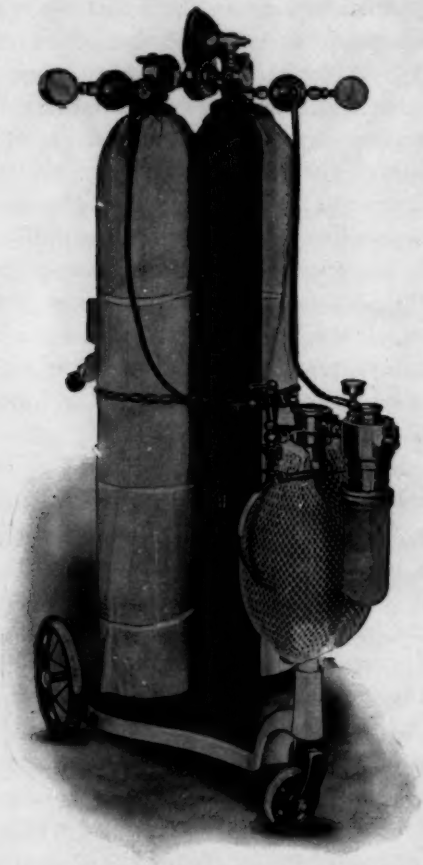


Fig. 2.—McKesson model G anesthetic machine. (Cut supplied through the courtesy of Dr. E. I. McKesson.)

post arising from the back of the machine. The cut from the instruction book of the McKesson people should make the layout of the machine reasonably clear.

The important points about the construction of the apparatus are: 1. There is sure metallic connection between all parts of the metal carriage and the important parts mounted on the head of the machine clear out to the outlet to which the hose attaches. 2. The tanks are

grounded to the carriage by means of supporting chains attached to the posterior post. It should be noted that the insulating cloth tank covers shown in figure 3 are not used in this hospital. To be doubly sure that the tanks are electrically connected to the frame of the machine, the rubber mat on which the tanks rest should be removed or holes pierced in it so that the tanks may stand on the frame itself from which insulating paint has been removed. 3. The entire apparatus is insulated from the floor by means of the rubber tires with which the machine is provided. 4. The breathing tubes are made of rubber in which is embedded a spiral conductor running the full length of the hose and making electrical connection with the metal collars at both ends. If a metal facepiece is employed, there exists an uninterrupted metal circuit from the metal rims of the wheels out to the facepiece. This means that if a charge is imparted to any metal part of the apparatus, all the metal on the machine receives equally the charge, and no sparks between metal parts can occur. It also means that the gas machine constitutes a conductor of considerable size so that, as observed in our theoretical discussion, the stage is set for a lusty spark should a charged body be brought near.

It should be pointed out that simply grounding a machine, as we have described, is worse than not grounding it at all if, at the same time, precautions are not taken to prevent charges from being brought near the apparatus. The effect of grounding the machine is to cause it to act like a conductor of infinite size, thereby making possible a static spark of maximum size. This matter is of extreme importance, as we find many users of gas apparatus grounding their machines in the belief that they have found the panacea that eliminates the danger of static discharges. Even at the risk of repetition, let us be sure that what is accomplished and what is not accomplished by simply grounding a gas machine is understood.

The usual practice is to run a copper wire from some point of the machine to a water pipe in the operating room. This accomplishes one thing. As long as the ground connection is intact, it maintains all the metal parts on the machine at the same potential as the ground, i. e., a zero potential. For this reason, no charges can be built up on the metal parts of the machine by passage of gases through certain parts of it or by other means acting within the apparatus. To this extent, grounding a machine may be desirable, but, as will be shown later, the production of charges on the metal parts of the machine by causes acting within the apparatus are of minor importance. Suppose a nurse comes rushing into the operating room carrying a high static charge with her. The grounded gas apparatus offers the best means for her

to get rid of the charge most rapidly and completely (like the radiator of our childhood days), so she proceeds to touch some part of the anesthetic equipment (or the table or patient, perhaps also grounded). Unfortunately the spark jumps quicker than her finger can move and before she touches any metal part, the spark has occurred, and perhaps an accident. Obviously, grounding of the anesthetic equipment, table and patient is worse than useless in preventing sparks due to charges that are generated outside and are brought into the danger zone. We will describe later the methods that have been used to eliminate these exogenous charges in other institutions.

STATIC CHARGES PRODUCED BY MOVEMENT OF THE GAS MACHINE

We will now describe the different parts of the apparatus and the things occurring in the operation that might conceivably lead to the production of charges on the gas machine.

Movement of the entire apparatus from the anesthetic room into the operating room or along the corridor facing the operating rooms may lead to the production of charges on the rubber tires of the machine. Equal and opposite charges are, of course, left on the floor over which the machine passes. This could not occur on floors that are damp or that are composed of a substance like the tires (i. e., made of rubber), but on dry linoleum, wood or tile floors charges are extremely likely to occur. The amount or the intensity of the charge that can collect on the tires will depend on the amount of actual friction that occurs between the tires and the floor and the excellence of the insulating qualities of the rubber in the tires. Obviously, sliding a machine sideways will lead to greater charges than simply rolling the machine, and damp weather or water on the floor will entirely prevent the accumulation of charges on the tires. It should be understood that charges produced by movements of the machine generate charges only at the points of contact of the tires with the floor, that is, at the periphery of the tires. If the charges remain in these locations they are of less importance than if they spread over the machine, although it must be remembered that even an extremely high charge on the tires would, when distributed over the entire metal parts of the machine, be greatly decreased in intensity. If the tires are excellent insulators—which is another way of saying if they are dry—the charges will be confined to the tread of the tires. If some leakage can occur, part of the charge will be lost on the floor and part will leak onto the machine.

A way in which static charges existing at the periphery of the tires may be of importance is by producing induced charges on the interconnected metallic elements of the machine. As we have described, a

negative charge existing on the tires would cause a positive charge to be induced at the nearest point of the conductor, which is here the metal rims of the wheels, and an equal negative charge to appear at the opposite end of the conductor, which would, of course, be at the tops of the tanks and on the metal facepiece. A person or conductor touching these points might cause a spark to occur. However, it must be remembered that the area over which these induced charges would exist is a large one and that an extremely intense charge on the tires would be required to induce negative charges of consequence on the tanks or the facepiece.

Herb,[8] and Allen and Murray [14] have reported explosions that have occurred while the gas machine was being moved from the anesthetic room. Nitrous oxide-oxygen and ether were in use when the accident took place in Chicago, whereas ethylene was being used in Detroit. In each of these instances it is difficult to attribute the trouble to charges that had accumulated by movement of the machine. Too many exogenous sources of static are present, the importance of which are definitely established.

Although it seems that explosion from charges generated by movement of the anesthetic apparatus can be regarded as unlikely, such a source of danger must be kept in mind. On several occasions when a gold leaf electroscope was used, we found appreciable charges on the tires of machines that had just been wheeled from the anesthetic rooms across the hall into an operating room. It was also found that these charges immediately vanished when the machine passed over a portion of the floor that had been recently mopped. In eliminating danger from this source, then, one must remember that a little moisture will prevent charges from being generated or from remaining on the tires. Particular attention should be paid to this matter in dry cold weather when the machine has to be transported for some distance.

STATIC CHARGES PRODUCED BY MOVEMENT OF ANESTHETIC GASES IN THE MACHINE

We will now discuss the problem of charges that are produced on the machine itself when the apparatus is in operation. We have said that charges are produced only when a certain amount of energy is expended as friction between unlike substances. One is forced to conclude that the possibilities for generation of static are decidedly limited. The only conceivable sources of charge on or within the apparatus are due to the passage of gases through the system or to movement of certain parts of the machine directly caused by the gaseous movement. It is plain that in only two places on the machine can such movement occur.

14. Allen, C. I., and Murray, M.: Surg. Gynec. Obst. **44**:690 (May) 1927.

They are, of course, the rubber supply bags (with the automatic shut-off valve mechanism) and the rebreathing bag, provided that rebreathing is being employed. We will first discuss the possibility of charges being generated by movement of the anesthetic gases themselves.

Luckhardt [15] pointed out the ability of gaseous mixtures flowing through an insulating tube to produce gradually static charges thereon. It is also known that liquids (not aqueous) flowing through nozzles or constricted orifices may lead to the generation of dangerous collections of static electricity on the nozzle or in an insulated tank into which the fluid falls. Gasoline has been exploded by charges originating in this way.[16] Furthermore, jets of steam passing out through small orifices from high pressure boilers or steam lines may carry a high charge. This steam striking a conducting object may impart the charge to it, so that many strange and perhaps dangerous phenomena may be directly traceable to an innocent appearing steam jet. It is interesting in this connection to recall that some of the early machines for the generation of large static charges used the principle of multiple jets of steam striking on an insulated conductor from which large charges could be obtained.

There exist two places on the gas machine where one might expect sizable charges to be generated by gaseous friction. The first location is at the reducing valve attached to the top of the gas tanks and the second is at the automatic shut-off valves located on the head of the machine. The reducing valve operates under a great pressure: 1,000 pounds (453.6 Kg.) per square inch, or more, on one side, and a much lower pressure, from 40 to 60 pounds (18 to 27 Kg.) per square inch, on the other side. Obviously the pressure gradient within the valve is tremendous, and within the highly constricted space through which the gas must pass, its velocity must be very great. One often has evidence of the great amount of decompression which occurs within the valve in frost which collects outside it. Why then does one not find large charges produced at this point? The answer to this question must be found in the construction of the valve and its housing. If a jet of gas were released to the outside from an ethylene tank, for example, with no intermediate valve, it might well be highly charged and impart its charge to any conductor on which it impinged. However, the expansion of the gas occurs within a closed chamber that is made entirely of metal. Therefore, even if charges are produced at the point in the valve where the gases undergo decompression, the charges tend to

15. Luckhardt, A. B.: Potential Dangers Attendant on Ethylene-Oxygen Anesthesia, J. A. M. A. **82**:1603 (May 17) 1924.

16. Standard Handbook for Electrical Engineers, ed. 5, New York, McGraw Hill Book Company, 1922, p. 1850.

be lost immediately because the charged particles of gas are attracted to the metal wall of the valve where any electrons that might have been displaced by the friction incident to the decompression process are free to return to the molecules from which they were removed. The whole point of the matter is this: Friction between gas and metal of valve may lead to charges on both. These charges must be opposite and equal; and following decompression, opportunity for neutralization of the charges is provided especially because the charged atoms of the gas are attracted to the oppositely charged metal. Let us emphasize here that should a charge be generated at the reducing valve it would appear diffusely spread over the metal parts of the machine, for we have shown that all these parts are electrically connected.

What has been said concerning generation of charges at the reducing valve applies equally to the automatic shut-off valves. It is clear, then, that if our ideas are correct, little danger from generation of charges by the gases passing through the metal parts of the machine can be anticipated. Tests with the electroscope on a considerable number of machines in operation have failed to reveal the slightest charge on the metal parts of the apparatus. It should be pointed out, however, that in operation the anesthetist is connected to the metal of the machine by her fingers on the facepiece and that the patient is also in the circuit as a result of the same contact. The anesthetist by her contact with the floor or the patient through the table may provide a path of leakage for charges originating on the machine. However this may be, it is evident that even under the system of operation in many hospitals, one involving no precautions for the elimination of sparks, charges on the metal of the machine are either not produced or are satisfactorily taken care of. Grounding the machine to a metal floor will absolutely eliminate danger from charges arising on the metal parts of the equipment.

Gases from the tanks pass down to the head of the machine through rubber tubes; then from the automatic valves into the large supply bags, from which they pass to the mixing chamber. From the mixing chamber the gaseous mixture, which may now be explosive, either passes directly out through the ether attachment and rubber hose to the patient or may be shunted through the rebreather before entering the hose. As we have pointed out, sparks occurring inside the machine cannot lead to explosion at points in the apparatus before mixture of the anesthetic gas with oxygen occurs. Thus explosion in the tubes leading from the tanks to the supply bags or in the bags themselves cannot take place, providing the machine is operating properly. This provision is added because it is possible, should the check valve at the bottom of the mixing chamber fail to seat perfectly, for contamination of gas in one supply bag from gas in the other to occur.

If by this means ethylene should leak into the oxygen bag the resulting mixture might be highly explosive. It should be stated here that the probability of the check valve not properly seating is not great, particularly if it is occasionally removed and inspected to see that it is clean and dry.

We must now conclude our discussion of charges that may arise by passage of the gases through the machine by considering the situation presented in the nonconducting portions of the apparatus.

We cannot say definitely that charges are not produced by passage of the gases through the rubber hoses employed. If any do exist they are located on the inside superficial portions of the rubber, and as the tubes are of uniform bore, one would expect the charge to be uniformly distributed along its walls. This being true, sparks could not occur between adjacent portions of the inner surface and, furthermore, electrified atoms of gas would be attracted to the charged rubber located more distally, so that conditions for neutralization of the charges, to a certain extent at least, would be provided. It should also be remembered that the velocity of the gases, particularly in the large breathing hose, is not extremely great, and one finds difficulty in understanding how friction sufficient to produce appreciable charge can result.

If the small hoses connecting the tanks with the supply bags receive a charge, it must be insignificant, since a number of tests with the electroscope have failed to reveal any static on these parts. The same is true of the larger breathing hose. Static charges occurring in the small hoses are of no importance as far as the danger of explosion in them is concerned, for obvious reasons. The opposite state of affairs prevails with regard to the breathing tube, so that precautions must be taken to prevent charges therein, no matter how vague the possibility for the existence of such charges may seem to be.

To be certain that the charges do not collect within the breathing hose, the entire inside of the hose must be made of conducting material electrically connected at the two ends to the metal collars one of which slips over the attachment on the ether chamber and the other onto the metal facepiece. The tubes that are supplied with the McKesson machine are constructed with a closely wound spiral conductor embedded in the rubber on the inside of the tube. If this conductor is in actual contact with the gases throughout the inner surface of the hose and makes satisfactory electrical contact with the metal end-pieces, the tube is safe as far as charges generated by passage of gases within is concerned. It should be pointed out that a spiral conductor deeply embedded in the substance of the rubber hose and not projecting into the lumen of the tube is powerless to prevent accumulation of charges on the inner rubber surface. It is not only useless in preventing inside charges, but it may provide a source of static spark since, as has been shown, a conductor

in close proximity to a charged body has charges induced on it and the insulating medium between them may rupture leading to a spark.

We have shown that the passage of the gases themselves through various parts of the machine is certainly not important as a source of charge within the machine. Explosive mixtures exist only in the mixing chamber and from there outward. Sparks cannot occur in the mixing chamber because it is entirely of metal interconnected with the rest of the machine. The same argument applies to the ether attachment and the rebreather (with an important exception to be considered later). The hose, however, may contain an explosive gaseous mixture and at the same time present the fuse necessary to set the mixture off. It is important to be sure that the hose is designed so that charges cannot collect within its lumen.

CHARGES PRODUCED BY MOVEMENTS OF RUBBER REBREATHER OR SUPPLY BAGS

Gases passing through the anesthetic equipment can cause charges to arise not only because of friction of the gases themselves on certain parts but also indirectly by causing motion and hence friction between adjacent structures. The supply bags are periodically distended and deflated during the operation of the apparatus, and friction occurs between the rubber bag and the coarse mesh that surrounds it and between the bag and the metal arm attached to the mechanism of the shut-off valve. When rebreathing is employed, the rubber rebreathing bag expands and contracts in synchronism with the patient's respiration, so that friction occurs between the rubber bag and the bottom of the metal chamber. The problem presented by the rebreather will be considered first.

The rebreathing chamber is a cylindric metal shell about 8 inches (20.3 cm.) long terminating in a rounded bottom in which several good-sized holes are punched. The upper end of the chamber is screwed into the under surface of the ether attachment. To vary the amount of rebreathing and to make the system a closed one, a cylindric rubber bag, fastened on a movable metal ring encircling the metal shell, is fitted over the bottom of the device. A fine mesh net cover is provided for the rubber bag. As these bags are made of thin rubber they deteriorate after some time and have to be replaced. As a matter of convenience in this hospital, as the original bags with the net wore out, rubber gloves were substituted for them. Thus practically all the machines now in use have been equipped with a bare rubber glove which serves as the extremity of the rebreathing space.

As McKesson [17] pointed out, respiratory gases never reach this end of the rebreathing system since its capacity is far too great. For this

17. McKesson: Instruction Book for Model G Apparatus.

reason, the rubber glove is always perfectly dry and contains only mixtures of the anesthetic gases and ether vapor, if the latter is being used. It is hardly necessary to say that at some time during ethylene anesthesia, highly explosive mixtures of gases may exist here and, furthermore, that with the administration of nitrous oxide-oxygen and ether vapor the same may be true.

Observation of the rubber glove when rebreathing is being used proves that a certain amount of friction between the rubber and the rebreather is always present. During expiration the glove is ballooned out so that it makes scanty contact with the metal shell but during inspiration the rubber is drawn tightly against the sides of the chamber and the fingers of the glove against the rounded bottom. The amount of friction at this point varies with the depth of the patient's respiration and the amount of rebreathing employed. Theoretically, in this arrangement the stage is set for the generation of charges. By actual tests with the electroscope, charges, often of great intensity, are regularly found on this rubber glove, especially toward its lower part. As a matter of fact, this glove was the only place on the machine where, during its operation, charges could be demonstrated.

It is a matter of great interest to us to find how well the actual electrostatic observations on the machine coincided with the theoretical possibilities. For example, friction of rubber on metal is supposed to result in a negative charge on the former and a positive charge on the latter. The electroscope proved the charge on the glove to be, in fact, negative. A positive charge of equal magnitude must, of necessity, be generated on the metal rebreathing cylinder, but since the rebreather is only a small part of the interconnected metal parts of the machine over which this charge must be distributed, no positive charge can be demonstrated. Moreover, another important fact must be remembered. Contact between the metal anywhere on the apparatus and a person or other conductor will further distribute the positive charge. As a matter of fact, during an operation the anesthetist and patient are grounded to the metallic elements of the apparatus, thus receiving their shares of the charge and offering further paths for its dissemination. Conditions on the rebreathing glove are much different. Because of the insulating nature of the rubber, the negative charge on the glove cannot be conducted away. The entire charge is bound to remain in situ, and its intensity is correspondingly great. Thus, by actual test, high negative charges are found on the rebreathing glove and nothing on the metal of the machine, although equal positive charges must have existed thereon, momentarily at least.

Tests of the magnitude of the charges produced on the rubber glove by means of an electroscope giving quantitative readings have shown

that potentials of several hundred volts are common. The length of time over which charges may persist on these gloves is considerable. Four machines in the anesthetic work-room were examined for charges at 2 p. m. None of these machines had been used since noon and some had been idle for three hours. High charges were found on the rubber glove on three of the four. The one machine on which no charge was present was equipped with the original McKesson rebreathing bag with the net covering.

The presence of a mesh cover over the rebreathing bag has been thought by some to be a source of danger by virtue of friction between the two leading to static charge. Our experience does not seem to confirm this belief. In fact, the one machine equipped with the original bag and net is the only one on which charges have not been found during the course of an anesthesia. To explain this, it seems that one must assume that friction of cloth against rubber is not so potent a source of charge as friction of rubber against metal. Of the latter, there can be no doubt.

An extremely important point in connection with a charge arising on the rebreathing bag is its location. Is the charge inside or outside of the bag? Obviously, charges produced on the inner rubber surface and leading to spark within the bag are much more serious than external charges and sparks on the outside. It should be plain that friction of a rubber bag with a net cover can result only in charges accumulating on the outside of the bag; for this reason, even should charges between the netting and the bag be evident, we cannot consider their presence to be extremely dangerous.

In view of the foregoing discussion, it can be seen that the charges generated by friction of the rubber glove on the metal rebreather are highly dangerous. The charge on the glove is on its inner surface, and as parts of the rubber are continually being drawn in close proximity to the metal shell, sparks are likely to occur between the two. If the mixture in the rebreather happens to be an explosive one at the time the spark occurs and the spark liberates sufficient heat to ignite it, a tragedy is inevitable. Many facts lead us to believe that such a mechanism was responsible for a fatal explosion which occurred in the maternity wards of the University Hospital several years ago.

It is evident that the rebreathing bag, as it is being used in this hospital at the present time, constitutes a real point of danger. The fact that ethylene is not being employed does not eliminate the hazard, for ether vapor and oxygen may, in proper proportions, form highly explosive mixtures. This is a serious defect in the use of the machines which must be corrected even though the further use of ethylene is not contemplated.

The obvious method of preventing accumulation of charge on the rebreathing bags is to so modify them as to make them conductors of electricity. If, by some method of manufacture or some type of treatment, the rubber could be changed from an insulator into a conductor the problem would be solved. Some work has been done along these lines in the preparation of a leaded rubber bag. For some reason, however, this product has not appeared on the market. The use of water in the bags does a great deal to eliminate charges which might otherwise collect on the glove. The routine use of this method for prevention of charges is open, however, to two objections: 1. It does not seem to do the job. It must be remembered that the gaseous current through the rebreather may be very rapid and that water placed in the glove evaporates rapidly. It has been found that when the water becomes low in the fingers of the glove, upper portions of the rubber may be dry and possess slight charge. 2. It does not seem wise, at this important point, to rely for protection on a bit of routine which, after months of uneventful operation, may slip and result in a dry glove at the time when protection is most needed. Even with a staff of highly trained anesthetists, such as we possess in this hospital, it seems unwise to have the safety of the patient depend on the personal factor. Particularly is this true if other better means can be devised.

Prof. N. H. Williams, of the Physics Department of the University of Michigan, has suggested the use of a manometer or U-tube containing fluid to replace the rubber bag. In order that such a device may be used to measure the tidal air, as is done when the bag is employed, the layout shown in figure 3 should prove satisfactory.

As can be seen by inspection of the drawing, the rubber bag is replaced by a U-tube made of sheet metal. The proximal arm of the tube is securely soldered to the metal ring which slides over the rebreathing chamber. A liquid—water, glycerin or mercury, depending on the terminal resistance desired—is placed in the U-tube. Obviously, air forced into the rebreather under pressure will raise the liquid in the distal arm until a pressure balance is secured.

To measure the tidal air with the manometer device, one must first slide the U-tube up as high as it will go by moving the ring attached to the proximal arm up the rebreather. The fluid level in the manometer is then adjusted so that it lies just at the convex bottom of the rebreathing chamber. The tidal air is found by sliding the U-tube down until the correct volume is obtained, using the calibration on the rebreather. When the correct point is reached, that is, when the volume of expired air just fills the space between the bottom of the rebreather and the fluid level, the liquid in the manometer, as seen through the glass window, will oscillate perceptibly at each respiration. During this process the

expiratory valve on the facepiece must be adjusted so that no air escapes from it. This device may be open to criticism on the ground that it is rather bulky and cumbersome. On the other hand, it would eliminate the danger so inherent in the use of a rubber bag. Moreover it must be remembered that the manometric device need never be replaced and requires nothing but an occasional readjustment of the fluid level.

A means for avoiding charges on the rebreather which may prove to be valuable is the use of a radioactive substance in the bag. It is well known that radioactive substances, by the emission of alpha, beta or gamma rays, cause ionization of the surrounding air so that the air itself may become a conductor of electricity. The rebreathing bag is not large and it seems that the use of some ionizing material within it

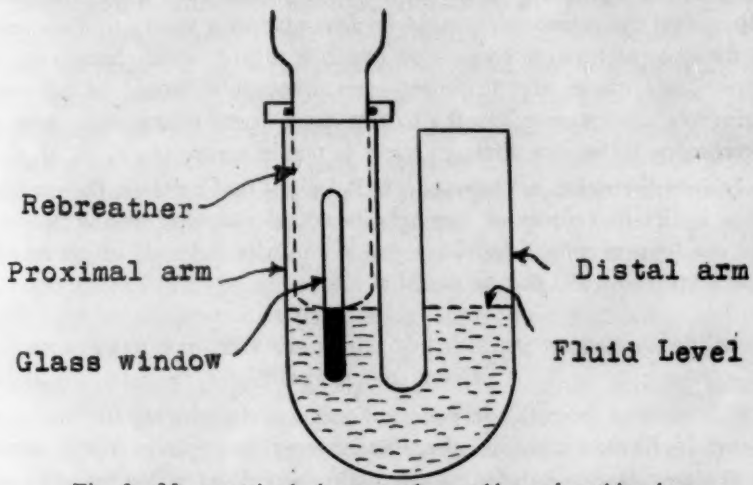


Fig. 3.—Manometric device to replace rubber rebreathing bag.

might so alter the character of the air within and around the rubber as to prevent the accumulation of charges thereon. Because of its simplicity, a trial of this method would seem to be advisable.

A considerable amount of space has been devoted to the discussion of charges on the rebreather because it is felt that, under the present operating conditions, danger is always present at this point. It should be strongly emphasized that an explosion in the rebreather is very likely to be fatal to the patient because the explosion travels in both directions from the point of ignition, back into the mixing chamber and out through the hose, even into the patient's air passages. Explosions occurring outside the machine are not so serious because the explosion is not confined to a closed space in direct communication with a person's vital organs.

Little has been said up to the present concerning charges existing on the large rubber supply bags. The danger of explosion within these

structures, except under very unlikely circumstances, as we have shown, is nil because they contain pure gases from the tanks. Charges existing within the bags can occur only by friction with the gases in them, since there is no source of metallic friction. This condition may arise but it is difficult to understand how charges so produced can be of any importance. Charges on the outside of the supply bags may arise either by friction between the rubber and the enclosing mesh or by friction between the metal arm, attached to the automatic shut-off valve, and the bag. Neither of these sources of static can be very important because, during the operation of the machines no charges have been found on the bags, with the electroscope. To be sure that charges do not collect on the bag it should be made of conducting material. Perhaps the use of a radioactive substance in or on the bag, should such a method prove valuable on the rebreather, would be advisable as a safety-first measure. At the present time, it seems that one can regard static charges on the supply bags as of minor importance, although it would be advisable during the cold winter months to test these parts occasionally with an electroscope to be sure that our view is not in error.

In our discussion we have covered charges that arise on the machine either by its movement or through its actual use, and means whereby they can be prevented. We have yet to consider methods of preventing static charge outside of the machine.

STATIC CHARGES ARISING OUTSIDE OF THE MACHINE AND THEIR PREVENTION

It is evident from the foregoing discussion that during the operation of the McKesson machine there exist very few places on it where charges may be generated or accumulate thereafter. Charges produced by forces operating outside of the anesthetic equipment are harder to classify. It is evident that they can be brought into the operating room by persons or objects entering it, or charges can be produced inside the room by movement of persons or friction between bodies therein. A system for prevention of charges within the danger zone must: (1) maintain the anesthetic equipment, patient, table and surrounding objects at the same electrical potential from the time the anesthesia is started until it is finished; (2) prevent persons or objects from approaching the operating table or entering the room while carrying charges; (3) include a realization of the fact that charges can be generated and can collect on insulating bodies near the patient if care is not exercised to prevent friction between such objects. For example, the patient's hair might be brushed by some unintentional movement and a charge thereby be generated.

To maintain the machine, patient, table, etc., at a constant potential, a floor of conducting material to which these objects are electrically joined, either by chains hanging from metal parts or by direct contact, must be provided. The installation at the Presbyterian Hospital in Chicago accomplishes this by a carefully designed floor which has been described by Herb.[3] Their problem was not extremely difficult because the anesthetic rooms in the Presbyterian Hospital are not separated from the operating rooms by a corridor as they are in the University Hospital. To take care of the situation here, metal sheeting, either of rustless steel or copper, should be placed on the floor of the anesthetic rooms. Directly continuous with this and extending across the hall and into the operating room should be laid more sheet metal of sufficient width to make sure that the machine remains on the metal on its passage to the operating room. Inside the operating room there should be laid a large sheet of the same material, of sufficient size to accomodate the entire surgical ensemble, with a liberal margin around the edges. This sheet should, of course, be directly connected with the one entering the operating room from the hall. It should be noted that the thickness or amount of metal in these sheets has nothing to do with their electrical efficiency. The sheets could be made of gold leaf and still serve satisfactorily as a conducting medium if they were mechanically able to stand the strain. In other words, the metal sheeting which is used need only be thick enough to possess satisfactory mechanical wearing qualities.

The machines should be equipped with metal chains fastened electrically to the frame of the machine and dragging an inch or so along the floor. Any other tables or stands used near the operating table should be similarly provided with chains so that all metal objects in the proximity of the table are kept at the same potential as the rest.

Nothing specific has been said about grounding the metal sheets which are to be laid on the floors. It should be evident that if all the sheets are properly joined and the chains are properly connected to the moveable equipment and make contact with the floor, all the metal parts must be at the same potential and sparks between metallic elements of the system are therefore impossible. This is true whether the floor is grounded or not. The only advantage that can be secured by grounding the system is the assurance that the whole outfit will always remain at the same potential, i. e., a zero potential. It is conceivable, but unlikely, that all the interconnected metal parts might achieve an appreciable positive or negative charge so that spark might occur on contact with a neutral body. We have shown that a large conducting system, particularly a grounded one, offers the best opportunity for sparks of maximal size when a charged body is brought near. This hardly con-

stitutes a sufficient reason for not grounding the floor, but a grounded metal floor presents some danger of short circuit or electric shock since one side of the electric power supply to the hospital is also grounded and the floor may thus act as one terminal of an electric circuit. Difficulty in this direction is especially apt to be found when electrical devices are used in the operating room. In view of the evidence, it would seem advisable to leave the interconnected metal floor ungrounded, at least until some evidence is forthcoming to show such a course unwise. It should be pointed out that grounding can be done easily at any time if it is thought advisable.

In an effort to prevent the entrance of any one to the operating room who might carry a charge, a strip of sheet metal should be carried up on the outside of the doors leading to it. These metal pieces must be electrically connected to the sheet metal flooring and so placed on the door that one must touch them when pushing the door open.

No matter how complete and thorough the means for interconnecting the metallic parts of the operating room equipment are, it must be remembered that charges may still be generated and exist on insulated portions of the apparatus or of other objects within the danger zone. Friction between nonconducting substances, particularly rubber and other materials, must be avoided. The practice of hastily stripping the cotton sheets off the protecting rubber one immediately following the removal of the patient from the operating table will invariably produce high charges on these sheets in suitable weather. The possibility of explosive mixtures of anesthetic gases existing at this time is, of course, not great but one cannot blindly rely on their absence. We have shown that friction of dry rubber on metal is a potent source of static charge. Rapid transfer of metal surgical instruments by persons wearing rubber gloves might lead to production of charges and to serious consequences, particularly at a time when the surgeon's gloves were dry and he was operating on the face or neck.

Friction against the rubber breathing tube might cause the production of charges on its outer surface. For this reason it is advisable to protect the rubber from trauma by winding its outer surface with a coarse mesh of copper wire, securely soldered at the ends of the tube to the metal terminals.

It should be emphasized that sparks occurring outside of the machine are important only because it is possible that explosive mixtures of the gases may be present at the point where the sparks occur. By using a metal floor continuous from the place where the anesthetic is started to the large conductor in the operating room to which the machine and other movable objects are electrically connected by dependent chains, and by using metal plates on all the operating doors so that a person

cannot enter without losing any charge that he might possess, a system is formed that largely eliminates the danger of exogenous charges. As we have shown, however, there exist other sources of static charge which, although they may seem unimportant, must be thought of. Many of these factors are not readily amenable to preventive treatment. In view of these conditions, it seems wise to recommend the use of an additional safeguard, namely, a device similar to the one used by Salzer [9] which will provide an exhaust pipe for the exhaled gases and prevent their accumulation in the operating room.

As we have mentioned, Salzer employs a metal sleeve which fits snugly over the expiratory valve of the McKesson facepiece. To the sleeve is attached a noncollapsible rubber tube which conveys the expired gases to the outside. Obviously, the rubber tube used for this purpose must be protected, as is the breathing hose, against static charges. A

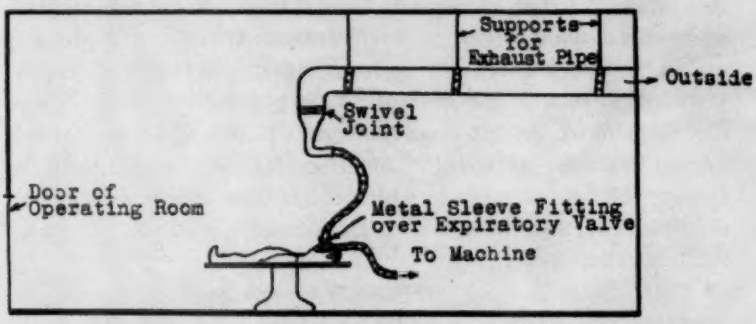


Fig. 4.—Arrangement, as suggested by Salzer, for removing expired gases from operating room.

piece of the breathing hose covered on the outside by a protective wire network would serve admirably for this purpose (fig. 4).

Before we conclude this section of the paper with a summary of the recommendations for the prevention of anesthetic gas explosions in this hospital, we should briefly consider some of the systems that are advocated by workers elsewhere.

We had the privilege of inspecting the installation which has been employed so successfully at the Presbyterian Hospital in Chicago. We were fortunate in being able to talk with Dr. Herb and with Mr. F. B. Johnson of the Commonwealth Edison Company who was in charge of the investigation carried out in the hospital, which led to the system now in use. The problem at the Presbyterian Hospital was limited to a method that would eliminate static charges arising outside of the anesthetic equipment. The McKesson machine is employed, but since rebreathing is not used, with ethylene at least, charges generated within

the machine were not considered of importance. It is unnecessary for us to describe the details of the system in Chicago for Dr. Herb has done that.[3] The installation provides a floor of conducting material, so that chains hanging from the machine and other movable objects can maintain all the metal parts of the equipment at the same electrical potential. The breathing hose is covered by a coarse network of wire; the patient is grounded to the table by an adequate conductor, and attempts to ground the outside of the rubber supply bags and the rebreathing bag to the frame of the machine by means of chains complete the salient points of the installation. An important feature of the floor is that conducting material exists from the point where the anesthetic is started clear through into the operating room and for a large distance around the table so that persons or objects brought near it may lose any charge they may carry before they arrive within the danger zone. It will be noted that this system is essentially the same as the one advised for this hospital, except that it is recognized that a thin sheet metal floor, which is as efficient electrically, will provide a cheap and satisfactory means to the same end, and that the floor which we recommended will not necessarily be grounded, as we believe such a course to have decided disadvantages. It should be mentioned that the system installed at the Presbyterian Hospital has proved its worth. During the four winters in which it has been in use, no explosions have occurred and tests have shown that charges were no longer present in the operating room.

The Gatch method of complete rebreathing employed by Poe[13] offers definite advantages, as much of the complexity of the anesthetic apparatus is done away with. The places where charges may be produced or accumulate are greatly reduced in number. Moreover, with this method the amount of gas escaping into the room is considerably reduced, so that explosions outside of the anesthetic apparatus are less likely. There are several reasons, however, why the adoption of such a system in this hospital does not seem feasible: 1. The method of administration is entirely different from the one now in use; its adoption would necessitate the purchase of new equipment and the disposal of a large amount of valuable apparatus—a somewhat expensive process. 2. We are forced to wonder whether the method is really as safe as figures would lead us to believe. Has not the freedom from accidents experienced by Dr. Poe and others been partly, at least, due to the more favorable climate in which they live? The use of a rubber bag of some size into which the gases are run and into which the patient breathes does not seem entirely to guarantee the user against explosion which, if it did occur, would be most serious to the patient. The presence of respiratory moisture and carbon dioxide in the bag

can hardly be relied on to prevent the existence of explosive mixtures therein nor can it be said that it is impossible for charges to be generated inside the bag.

Lewis and Boehm [18] described a method for preventing static spark on or near the machine by a method of combined metallic interconnection and grounding of the apparatus by means of wet towels, placed on the operating floor. The method seems to have settled the problem in their institution, but certain phases of the system seem rather inadequate to provide complete protection under all circumstances. In the first place, the simplicity of the method depends on the assumption that floors are grounded conductors. The tile floors ordinarily found in operating rooms may not be perfect insulators but they cannot be considered as good conductors of electricity. One must remember that the tiles are made of vitreous clay and are not unlike the material used for insulators on high tension transmission lines. It seems unlikely that, unless it is wet, the floor in their own hospital is able to conduct electricity and, hence, that it is grounded at all. In the second place, the use of moist cloths to transfer charges from the machine or patient to the floor is open to the objection that this means of protection introduces the "personal factor" into the situation. Through carelessness the cloths may not be moistened all over and the question is always to be asked, are the cloths still wet?

SUMMARY OF RECOMMENDATIONS FOR PREVENTION OF EXPLOSIONS

We believe that the following recommendations will provide a safe and workable system for the prevention of explosions during the use of ethylene oxygen or other anesthetic mixtures in this hospital.

General Measures.—1. Enforcement of strict regulations prohibiting the use of electrical equipment or any obvious source of heat in the vicinity of the anesthetic equipment.

2. The suggestions of Dr. McKesson for the technic of ethylene administration should, in general, be adhered to.

Measures to Prevent Explosions due to Static Spark Outside of the Machine.—1. Thin sheet metal (either copper, brass, or rustless steel) flooring forming a continuous pathway from the anesthetic rooms across the hall into the operating rooms where it should cover most of the floor space.

2. Chains electrically connected to and suspended from the anesthetic machines and other movable equipment so as to drag on the metal floor.

18. Lewis, W. B., and Boehm, E. F.: A Simple Method of Eliminating Danger of Explosion Due to Static Spark in Gas-Oxygen Apparatus, J. A. M. A. 84:1417 (May 9) 1925.

3. Inspection of breathing tubes to be sure that the spiral conductor embedded therein projects into the lumen of the tube and that it is electrically joined to the metal pieces at the extremities of the hose. The breathing tubes should be covered by a coarse network of copper wire which should likewise be soldered to its metal ends.

Metal plates, connected electrically to the metal floor should be placed on the outside of all doors leading into the operating rooms in such a way that one cannot open the door without touching the metal plate.

4. Connection between the patient and the metal frame of the operating table by means of a chain ending in a suitable piece of metal which must rest against his skin.

5. Holes cut in the rubber mat on the carriage of the machines and insulating paint removed from the metal platform at these points so that the tanks will be positively grounded to the frame of the apparatus.

6. A device such as the one used by Salzer should be installed to prevent the exhaust gases from being poured out into the operating rooms.

Measures to Prevent Explosions due to Static Spark Within the Machines.—1. Realizing that the bare rubber rebreathing bag as it is being used at the present time is very dangerous, we recommend (a) the trial of a small quantity of radioactive substance within the bag, and (b) if the foregoing method does not absolutely prevent charges from accumulating on the bag, replacement of the bag with the manometric device described.

2. Inspect the check valve admitting gases into the mixing chamber weekly to be sure that the valve and the valve seat are clean and dry.

Tests.—During the cold months of the year, carry out an occasional test with a gold leaf electroscope to be assured that the system is actually eliminating the danger from static electricity.

Prof. N. H. Williams of the Physics Department of the University of Michigan and Mrs. Laura Davis-Dunston, instructor in anesthesia and chief anesthetist at the University Hospital, helped in the preparation of this paper.

THE VOLUME OF BLOOD FLOW PER MINUTE THROUGH THE LUNGS FOLLOWING COLLAPSE OF ONE LUNG BY OCCLUSION OF ITS BRONCHUS

EXPERIMENTAL OBSERVATIONS *

RICHMOND L. MOORE, M.D.
NEW YORK

The effect of atelectasis [1] of a lung on the pulmonary blood circulation became particularly interesting with the introduction of pneumothorax in the treatment for certain types of pulmonary tuberculosis. The significance of this relationship has increased in importance with the development of thoracic surgery, because there are few, if any, surgical procedures within the thoracic cavity or involving the thoracic wall that do not affect the expansion of the lungs. In spite of many experimental studies, however, there is no uniform agreement as to the exact nature of the resulting circulatory changes. Different investigators have arrived at conclusions that are diametrically opposed to each other, and the present status of knowledge is at best confusing and nonconvincing.

The contradictory results are in part due to the fact that so far it has not been possible to determine by direct measurement the amount of blood that passes through a lung. Indirect methods offered the only means for the study of this problem, and the methods that have been used are varied and in some instances based on concepts now known to be erroneous.

Other causes for confusion can be traced to the several experimental procedures that have been used. Some writers have described the circulatory changes that accompany bronchial occlusion; others, the circulatory effects of a closed pneumothorax, and still others, the effects of an open pneumothorax. Whereas total or partial collapse of a lung may result from all of these procedures, the effect on the pulmonary

* Submitted for publication, May 2, 1930.

* From the laboratories of the Department of Surgery, College of Physicians and Surgeons, Columbia University, New York.

1. The term, "atelectasis," is used in this paper to denote diminished pulmonary expansion and not a state of complete collapse of the pulmonary alveoli. The term, "apneumatosis," suggested by Coryllos and Birnbaum (Obstructive Massive Atelectasis of the Lung; Arch. Surg. **16**:501 [Jan.] 1928), more accurately describes this latter condition.

circulation, theoretically at least, may be quite different, because the physiologic alterations associated with each of these conditions are not the same. When a primary bronchus is totally occluded, collapse of the corresponding lung results from absorption of the trapped gases. As the gases are absorbed, the lung shrinks and eventually comes to occupy a much smaller portion of the thoracic cavity. The corresponding half of the diaphragm moves upward, and the mediastinum is pulled over to fill up the additional space. The elevation of the diaphragm and the displacement of the mediastinum follow readily, because both are yielding structures. The thoracic wall, on the other hand, is rigid and cannot be sucked inward. Under these conditions, the lung collapses toward the wall of the chest, and the pressure that is developed in the pleural space on the side of collapse during inspiration is probably more negative (i. e., unless the mediastinum can move over far enough to equalize the pressure) than it was under normal conditions.

In closed pneumothorax and open pneumothorax the lung collapses toward the mediastinum, and both of these conditions are accompanied by a decrease in the negative intrathoracic pressure and a shifting of the mediastinum to the opposite side of the thorax. In this way serious impairment of the expansion of the "opposite" lung in the closed half of the chest may accompany the reduced expansion of the lung on the side of pneumothorax. Attention was directed to this effect on the opposite lung by Bruns [2] in 1912; more recently Graham [3] has proved it to be true by animal experimentation. This is an important consideration, because, as will be brought out later, some of the methods that have been used to estimate pulmonary blood flow are based on the assumption that in the presence of a unilateral open or closed pneumothorax the expansion of the contralateral lung is normal, or even increased.

In closed pneumothorax, the lung is compressed, and when the mediastinum is sufficiently rigid or sufficiently fixed by adhesions to prevent compression of the opposite lung, the intrapleural pressure on the side of pneumothorax can be raised to such a degree that it will exceed atmospheric pressure even at the height of inspiration. These are well known facts. I have repeated them here merely to illustrate the necessity of distinguishing between the effects of compression and atelectasis when studying the changes in the pulmonary circulation that accompany a closed pneumothorax.

---

2. Bruns, O.: Untersuchungen über den respiratorischen Gaswechsel bei Erkrankungen der Lunge und der luftzuführenden Wege, Deutsches Arch. f. klin. Med. **107**:468, 1912.

3. Graham, E. A., and Bell, R. D.: Open Pneumothorax: Its Relation to the Treatment of Empyema, Am. J. M. Sc. **156**:839, 1918.

These important physiologic changes must be kept in mind when one attempts to evaluate the experimental evidence that has been accumulated by others. If it is not known to what extent the expansion of a "collapsed" lung has been impaired, one cannot know accurately what is meant by the term "collapse." It is of equal importance to know also whether or not the expansion of the opposite lung has been affected. In general, it may be said that under normal conditions pulmonary expansion is dependent on the amount of negative intrathoracic pressure that is developed during inspiration. The importance, then, of changes in intrathoracic pressure cannot be overlooked.

The experiments of previous authors are not always specific in these important details. For some, "collapse" and "pneumothorax" are synonymous, and many overlook the effect of a closed or open pneumothorax on the expansion of the opposite lung.

Impressed primarily by the confusion that has arisen from the use of the term "collapse" in what might be called a qualitative sense, it occurred to me that a method which permitted quantitative measurement of the expansion and circulation of each lung separately would offer an excellent means for the study of the effect of atelectasis on pulmonary blood flow. In this communication, I am reporting the results of such a study. Complete collapse (apneumatosis) of one lung (either the right or left) has been produced in anesthetized dogs without restricting the breathing of the opposite lung, and the volume of pulmonary blood flow per minute through each lung separately has been measured before and after collapsing the lung. The results have consistently shown that the pulmonary circulation is strikingly decreased in a lung that has been rendered completely airless by occlusion of its bronchus. The knowledge and experience gained have led me to believe that the method can be used to study the pulmonary circulation in association with varying degrees of compression of the lung, and experiments of this character are now being considered.

HISTORICAL REVIEW

Lung Collapsed by Bronchial Occlusion.—Loewy and von Schrötter [4] introduced a lung catheter with a rubber balloon attached to one end into a branch bronchus or one main bronchus of man. By inflating the balloon, they were able to cut off completely the corresponding part of a lung, or a whole lung, from the rest of the bronchial tree. They found that a considerable portion of one lung could be deprived

4. Loewy, A., and von Schrötter, H.: Untersuchungen über die Blutcirkulation beim Menschen, Ztschr. f. exper. Path. u. Therap. **1**:197, 1905.

of its ventilation without necessarily causing a decrease in the oxygen content of the arterial blood. In these experiments they assumed that the blood from the unventilated lung would be venous and should cause an increased unsaturation of the arterial blood. The fact, however, that the saturation of the arterial blood was unchanged, or only slightly reduced, suggested that the blood flow through the unventilated lung tissue was decreased.

Hess [5] estimated the oxygen content of the arterial blood in a series of rabbits and then obstructed the primary bronchus of the left lung by introducing a tampon cannula and inflating a small rubber balloon which was attached to its end. Just as Loewy and von Schrötter had done before, he reasoned that after bronchial occlusion the arterial blood would be a mixture of venous blood (with low oxygen content) from the blocked lung and arterial blood (with high oxygen content) from the breathing lung. Hess assumed that under normal conditions the flow of blood through the left lung is one-third less than that through the right. Assuming further that the normal fraction of blood passed through the left lung after its bronchus was occluded, he calculated what the oxygen content of the arterial blood should be from the control arterial oxygen content [6] and from the oxygen content of the venous blood after blocking. He found that the oxygen content of the arterial blood was approximately what he estimated it to be, and concluded that occlusion of a primary bronchus does not destroy the circulation in the corresponding lung; that approximately as much blood goes through after occlusion as before. The animals breathed air in all experiments, and observations were not made later than from five to ten minutes after bronchial occlusion. Autopsy never revealed a lung that was completely collapsed.

Adams and Morris [7] noted a marked fall in the oxygen saturation of the arterial blood of dogs after occlusion of the main bronchus of the left lung. Administration of oxygen rapidly raised the arterial saturation to normal. This return of the arterial saturation to normal with one main bronchus occluded would not have been possible if blood were still circulating through the blocked lung. The observation suggested that the diminished saturation which followed bronchial occlusion

5. Hess, R.: Ueber die Durchblutung nicht atmender Lungengebiete, Deutsches Arch. f. klin. Med. **106**:478, 1912.

6. In this calculation the oxygen content of the blood from the breathing lung (right lung) is assumed to be the same as the control arterial oxygen content. The formula from which the calculation is made is given in the paragraph which describes the experiments of Weiss.

7. Adams, D. K., and Morris, N.: Anoxaemia and the Administration of Oxygen, J. Physiol. (Proc. Physiol. Soc.) **54**:102-104; 106-107, 1921.

was primarily due to insufficient oxygenation of the venous blood in the functioning lung (the animals were breathing air), and that the circulation in the occluded (collapsed) lung was severely impaired.

Le Blanc [8] estimated the oxygen content of the arterial and venous bloods before and after occlusion of one main bronchus and before and after the induction of a one-sided closed pneumothorax. In the first experiments, the bronchus was blocked by inflating a small rubber balloon which was attached to the end of a flexible catheter. Experiments were made on dogs, cats and rabbits during the inhalation of air. He found that shutting off one lung from breathing by occluding its bronchus was regularly followed by a significant fall in arterial oxygen content. Assuming that the blood which passed through the functioning lung was normally oxygenated, Le Blanc attributed the fall in oxygen content to the unarterialized blood from the blocked lung and concluded that the circulation in the blocked lung did not appreciably vary from the normal. Le Blanc stated, however, that at autopsy the lungs of these animals were always found to be inflated.

Andrus [9] produced collapse of the left lung in dogs by ligating the primary bronchus and studied the circulation through the lungs before and after ligation. The total volume of blood per minute was determined by measuring the carbon dioxide output per minute and dividing this figure by the difference between the carbon dioxide content of the venous and arterial bloods. The average total volume of blood per minute of nine animals before operation was found to be 2,350 cc. One-half hour after operation, the average total flow was calculated from the average pulse volume before operation (19.1 cc.) and the average pulse rate after operation (141) and found to be 2,680 cc., an increase of 14 per cent over the preoperative figure. One-half hour after operation, he found the average flow through the unblocked lung to be 1,830 cc., and subtracting this from the calculated total flow concluded that 850 cc., or only 31 per cent of the entire cardiac output, was flowing through the collapsed lung. Twenty-four hours later, he calculated a further decrease to 28 per cent and on the thirty-first day a decrease to 8 per cent.

---

8. Le Blanc, E.: Respiratorischer Gasaustausch und Lungendurchblutung unter normalen und krankhaften Zuständen der Atmungsorgane. Untersuchungen am arteriellen und venösen Blut von Mensch und Tier, Beitr. z. Klin. d. Tuberk. **1**:21, 1922.

9. Andrus, W. DeW.: Observations on the Cardiorespiratory Physiology Following the Collapse of One Lung by Bronchial Ligation, Arch. Surg. **10**:506 (Jan.) 1925.

Coryllos and Birnbaum [10] produced experimental obstructive atelectasis and also experimental pneumonia in dogs and studied the lungs both by roentgenologic and histologic examination after intrajugular injections of iodized oil and india ink. They found that the iodized oil penetrated into the finer branches of the pulmonary artery and rendered them clearly visible in the x-ray pictures, but did not pass through the capillaries to the left side of the heart. No differences were apparent in roentgenograms between the affected and healthy lobes, other than dilatation of those branches of the pulmonary artery that supplied the atelectatic and consolidated parts. The dilatation was attributed to obstruction in the capillaries.

The india ink was injected under similar experimental conditions, but, in contrast to the iodized oil, readily went past the arterioles and penetrated the capillaries of the alveolar walls. The ink, just as the oil, was found to be distributed throughout both the affected and normal lobes. Striking differences, however, were noted in the number, size and shape of the injected capillaries of the atelectatic and consolidated areas. Coryllos and Birnbaum related these to the degrees of collapse of the corresponding alveoli and concluded that the capillary circulation is impaired in both atelectasis and pneumonia, and that the impairment is "due to, and regulated by, the degree of collapse of the alveoli."

Bruns came to the same conclusion in 1912. In that year, in a discussion of the pulmonary circulation in breathing and atelectatic lungs,[11] he said:

> Es ist also der Beweis erbracht, dass die Durchblutung normal ausgedehnter Lungen grösser ist als die kollabierter atelektatischer Lungen, ebenso wie ja auch die Blutmenge, die sich in einem gegebenen Augenblick in ausgedehnten, atmenden Lungen befindet, nach meinen Untersuchungen grösser ist als die in kollabierten. Je intensiver der Lungenkollaps, je ausgesprochener die Luftleere der Alveolen, desto kleiner ist die Blutmenge und die Stromgeschwindigkeit. Die Art der Ausschaltung der Atemfunktion spielt keine Rolle.[12] (It has been proved that a greater amount of blood passes through normally distended lungs than through collapsed atelectatic ones; likewise, according to my studies the amount of blood present in the lungs at a given moment is greater when they are distended in respiration than when they are collapsed. The more intense the collapse of the lungs, the less is the amount of air in the alveoli, and the less is the amount of blood and its velocity. The type of exclusion of the respiratory function does not play a rôle.)

---

10. Coryllos, P. N., and Birnbaum, G. L.: The Circulation in the Compressed, Atelectatic and Pneumonic Lung (Pneumothorax-Apneumatosis-Pneumonia), Arch. Surg. **19**:1346 (Jan.) 1929.

11. Bruns, O.: Die Blutzirkulation in atmenden und atelektatischen Lungen, Deutsche med. Wchnschr. **39**:101, 1913.

12. Bruns' experiments are described in the section on "Lung Collapsed by Closed Pneumothorax," p. 231, this issue.

These studies of Coryllos and Birnbaum are interesting, but they do not give any information as regards the amount of blood that was circulating through the atelectatic and consolidated lobes during life. The histologic evidence cited, however, indicates that after death the atelectatic and consolidated areas contained less india ink than the expanded parts.

Lung Collapsed by Closed Pneumothorax.—Bruns [2] studied the oxygen and carbon dioxide contents of both the arterial and the venous blood of rabbits, first, with the pleural cavities intact, secondly, after the production of a one-sided open pneumothorax and thirdly, after converting the open pneumothorax into a closed pneumothorax. He noted that open pneumothorax was regularly accompanied by a decrease in the oxygen contents and an increase in the carbon dioxide contents, but that both of these values immediately returned almost to normal when the chest was closed. This occurred even though the expansion of the atelectatic lung on the side of pneumothorax did not return to normal for about three days. The recovery of these values when the lung on the side of pneumothorax was still collapsed indicated, Bruns thought, that the changes could not have resulted from an admixture of imperfectly aerated blood from the collapsed lung. He concluded that the pulmonary circulation in the collapsed lung (lung on side of pneumothorax) was considerably reduced, and attributed the decrease in oxygen content and the increase in carbon dioxide content to a deficient ventilation of the opposite lung. Bruns maintained that when an opening in the thoracic wall is sufficiently large, the expansion of the opposite lung is impaired by a moving over of the mediastinum during inspiration; furthermore, that the emptying of the opposite lung during expiration is also imperfect, because the mediastinum now absorbs some of the pressure that results from collapse of the thoracic wall and elevation of the diaphragm by shifting back again to the side of the pneumothorax.

In other experiments, Bruns [13] estimated separately the colorimetric value of the blood that is present in the right and left lungs of rabbits, first under normal conditions and then in the presence of a one-sided open or closed pneumothorax. He found that normally the amount of blood that is present in the right lung is to the amount in the left lung as 3:2. After the production of pneumothorax, the readings indicated consistently that a lung in the collapsed position contains less blood than it does in the position of natural expansion. Bruns also studied the

13. Bruns, O.: Ueber die Blutzirkulation in der atelektatischen Lunge, Deutsches Arch. f. klin. Med. **108**:469, 1912.

unit volume of blood flow through the lungs before and after the creation of a unilateral open, or closed, pneumothorax. He accomplished this by perfusing the lesser circulation with defibrinated blood through cannulas which had previously been tied in the pulmonary artery and left ventricle. The cardiac output was taken as an index of the amount of blood passing through the pulmonary vessels. The output of the left ventricle was always lower after pneumothorax had been produced.

Bruns [14] concluded that the manner in which collapse was produced made no difference. He applied his results with equal importance to atelectasis from pneumothorax, pleural effusions, pleural thickening and bronchial occlusion. He attributed the diminished circulation to an increased vascular resistance resulting from the winding and coiling of the pulmonary vessels and also to a possible vasoconstrictor effect.

Cloetta [15] recorded the pressure changes in the pulmonary artery, right ventricle and carotid artery during the different phases of respiration, in dogs, cats and rabbits. He controlled the expansion of one or both lungs artificially by negative pressure in a plethysmograph and observed a fall in carotid pressure and a rise in pulmonary pressure whenever the lungs were inflated beyond the first one fourth or one third of the inspiratory phase. These changes were accompanied by a decrease of the systolic pressure and an increase of the diastolic pressure within the right ventricle. Cloetta also estimated the amount of blood in the lungs during the different respiratory phases from microscopic sections, and found that the amount of blood was less in the expanded lung than in the collapsed lung. As a result of his studies, he concluded that the pulmonary circulation is least at the height of inspiration, much better during expiration and best at the beginning of inspiration. Cloetta attributed the impairment of the blood flow during the latter half of inspiration to a stretching and narrowing of the pulmonary vessels. During the first part of inspiration, he believed that both the length and the cross-section of the vessels were increased. It is worthy of note here that he attributed the favorable influence of pneumothorax and thoracoplasty in pulmonary tuberculosis to an optimal flow of blood in the compressed lung.

White and Gammon [16] studied the fate of particles of fat that were introduced intravenously into the pulmonary blood stream of rabbits

14. Bruns (footnotes 11 and 13).

15. Cloetta, M.: In welcher Respirationsphase ist die Lunge am besten durchblutet? Arch. f. exper. Path. u. Pharmakol. **70**:407, 1912.

16. White, W. C., and Gammon, A. M.: Some New Features of Interest about the Pulmonary Circulation and the Fate therein of Intravenously Introduced Fats, Tr. Nat. A. Prev. Tuberc., Tenth Annual Meeting, 1914, pp. 215-220.

after the production of a unilateral artificial pneumothorax. They found that all the fat, save a very little which lodged at the hilus of the lung on the side of pneumothorax, went into the opposite lung. They do not mention either the type or the severity of the pneumothorax that was created; nevertheless, the observation suggests that under the conditions of their experiments an unknown degree of collapse, or compression of a lung, was associated with a severe impairment or complete obliteration of its blood supply.

Brauer [17] maintained that the circulation through a lung that has been partially collapsed by pneumothorax, or thoracoplasty is slowed by stasis, and furthermore, that the increase in the blood flow through the opposite lung is slight.

Le Blanc [8] found that a unilateral closed pneumothorax was regularly followed by only a slight oxygen deficit of the arterial blood, and concluded from this observation that the circulation in a lung that has been compressed by pneumothorax is considerably reduced.

Dock and Harrison [18] studied the flow of blood through the lungs of rabbits before and after the production of a one-sided closed pneumothorax. Studies were made at intervals of from one hour to five days after the introduction of air into the right pleural cavity. The volume of flow of blood per minute was estimated from measurements of oxygen consumption and oxygen content of arterial and venous blood samples. The proportion of blood passing through the lung on the side of pneumothorax was calculated by subtracting the blood flow through the breathing lung from the total blood flow. Dock and Harrison found that immediately (from one to two hours) after the production of pneumothorax the flow through the "collapsed" lung was slightly more than half of the total flow, whereas after from three to five days it was less than one fifth of the total flow.

In experiments on rabbits during the inhalation of oxygen, Weiss [19] computed the relative amounts of pulmonary circulation through the two lungs after a unilateral closed, and after an open pneumothorax. The computation was based on the obvious assumption that arterial blood from the carotid artery is a mixture of blood from both the right and the left lungs. Weiss reasoned that the character of the blood returning to the left side of the heart from the lung on the side of

17. Brauer, L.: Ueber die operative Behandlung der Lungentuberkulose, Deutsche med. Wchnschr. **47**:548, 1921.

18. Dock, W., and Harrison, T. R.: The Blood Flow Through the Lungs in Experimental Pneumothorax, Am. Rev. Tuberc. **10**:534, 1925.

19. Weiss, R.: Ueber die Durchblutung der Kollapslunge beim experimentellen Pneumothorax, Ztschr. f. d. ges. exper. Med. **53**:138, 1926.

pneumothorax must necessarily be venous, and when the saturation of the mixed arterial blood did not fall below 90 per cent, he assumed that the decrease in saturation was due entirely to the venous blood that was returning from the "collapsed" side, and that the circulation through the "collapsed" lung was very small. Weiss did not think it conceivable that a high degree of saturation of the arterial blood could be maintained if venous blood was coming from the "collapsed" lung in large amounts. When the saturation of the arterial blood fell below 90 per cent, he assumed that the expansion of the opposite lung had been affected, and that the decrease was due, not only to venous blood from the "collapsed" lung on the side of pneumothorax, but also to venous blood from the unaerated portions of the lung on the opposite side. Weiss calculated the value of X and Y from the two formulas: $X + Y = 100$ and $AX + VY = M(X + Y)$, in which A is the per cent of saturation of the arterial blood from the breathing lung; V the per cent of saturation of the venous blood from the collapsed lung; M the per cent of saturation of the mixed blood from the carotid; X the per cent of total pulmonary circulation through the breathing lung, and Y the per cent of total pulmonary circulation through the collapsed lung. The value of A was assumed to be 102.1 per cent. Weiss found that the pulmonary circulation through the lung on the side of pneumothorax was strikingly small in rabbits. In several experiments it was less than 12 per cent of the total pulmonary circulation.

In contrast to these results, Weiss found in similar experiments on dogs that considerable amounts of blood (up to 70 per cent of the total cardiac output) may pass through the nonaerated parts of the lungs. This calculated result was due to the marked decrease in the percentage of saturation of the arterial blood which followed production of a pneumothorax in these animals. As already indicated, Weiss attributed this conspicuous decrease to a shifting of the mediastinum and partial callapse of the opposite lung.

In several experiments, Weiss measured the oxygen absorption of his animals in addition to the arterial and venous oxygen contents and calculated the cardiac output per minute according to the Fick formula. He found that in the majority of instances the cardiac output was diminished.

Peters and Wolley [20] injected carbon particles (lamp black) into a vein of the ear of a rabbit after the induction of artificial pneumothorax. They found that the lung on the side of the pneumothorax filtered out

20. Peters and Wolley, cited from Lilienthal, H.: Thoracic Surgery, Philadelphia, W. B. Saunders Company, 1926, vol. 2, p. 325.

a smaller quantity of carbon, and took this as evidence that a smaller amount of blood circulated through it than through the opposite lung.

In more recent studies on anesthetized dogs, Andrus and Wilson [21] found that a closed pneumothorax of moderate size was accompanied by a 25 per cent increase in the amount of blood circulating through the lungs per minute, whereas pneumothorax of a larger size reduced the circulation through the lungs to 26.5 per cent below its resting value. The amount of blood flowing through the lungs per minute was calculated by dividing the oxygen consumption per minute by the difference between the arterial and venous oxygen contents.

Lung Collapse by Open Pneumothorax.—Sackur [22] produced an open pneumothorax on one side in five rabbits and one dog and measured the oxygen content of the arterial blood before and after the pleura was opened. He found that the oxygen content of the arterial blood decreased 50 per cent after creation of a right-sided pneumothorax. The change after a left-sided pneumothorax was similar, but less pronounced. Assuming that the ventilation of the lung opposite to the side of pneumothorax was increased, and furthermore, that as a result of this increased ventilation the blood passing through the opposite lung was better oxygenated, Sackur calculated the flow of blood through the lung on the side of pneumothorax, and found that it was always greater than the normal figure.

Sauerbruch [23] also expressed the belief that in a one-sided open pneumothorax the inactive, collapsed lung receives the greater part of the pulmonary blood supply. Reasoning purely on physical grounds, he stated that when the pressure in one pleural cavity approaches the pressure within the corresponding lung, the pulmonary vessels dilate and offer less resistance to the passage of blood. The blood from the collapsed lung is returned to the left side of the heart with a low oxygen content, and anoxemia and dyspnea result.

Mention has already been made of the experiments of Bruns [24] and Weiss.[19] In contrast to Sackur, both of these authors concluded from their experiments on rabbits that the amount of circulation in a lung collapsed by open pneumothorax was considerably reduced.

---

21. Andrus, W. DeW., and Wilson, J. D.: The Effects of Closed Pneumothorax and Phrenicotomy on the Cardiorespiratory Function, Arch. Surg. **19**:1205 (Dec.) 1929.

22. Sackur: Zur Lehre vom Pneumothorax, Ztschr. f. klin. Med. **29**:25, 1896; Weiteres zur Lehre vom Pneumothorax, Virchows Arch. f. path. Anat. **150**:151, 1897.

23. Sauerbruch, F.: Zur Pathologie des offenen Pneumothorax und die Grundlagen meines Verfahrens zu seiner Ausschaltung, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **13**:399, 1904.

24. Bruns (footnotes 2 and 13).

Yates,[25] who has given much thought to this subject, said that "maximum unit volumes of blood are delivered to unit volumes of lung when the lung is in a position of incomplete deflation corresponding to reduction but not to abolition of intrapleural negative pressures."[26] He quoted the experiments of Cloetta in support of this opinion, and expressed the belief that one of the important factors in the therapeutic value of artificial pneumothorax and phrenemphraxis is the improved circulation (pulmonary) in the affected lung. Yates attributed this hypothetic improvement of the circulation to a decrease in the peripheral intravascular resistance, which allows the heart to propel more blood.

Willy Meyer,[27] in agreement with Yates, also accepts the evidence of Cloetta as probably indicating a hyperemia of the collapsed lung.

Torek,[28] on the other hand, expressed the belief that the greatest amount of blood is to be found in the expanded lung, less in the collapsed lung and least in the compressed lung. He said that the negative pressure which is responsible for expansion of the lung sucks in blood from the right side of the heart, and that when it is increased, as during inspiration, there is a consequent increase in the amount of blood sucked in; whereas when the negative pressure is abolished, as it is during expiration, the blood is driven out.

Eggers and Kernan,[29] commenting on the virtues of artificial pneumothorax in certain well selected cases of acute pulmonary suppuration, stated that there is less circulation in a collapsed lung than in an expanded one "owing to collapse of the alveoli with corresponding diminution in the size of the capillaries."

METHOD

Dogs were used. They were anesthestized by the intravenous injection of barbital sodium, which was dissolved in physiologic solution of sodium chloride and given in the proportion of 0.3 Gm. drug per kilogram of body weight. The preparation of the animals was similar to that originally reported by Churchill and Agassiz[30] and recently used by the author in an experimental study of the vagal

25. Yates, J. L.: Rationale of Operations Helpful in Promoting Recoveries from Pulmonary Tuberculosis, Arch. Surg. **14**:369 (Jan.) 1927; in discussion of papers on "Acute and Chronic Nontuberculous Bronchopulmonary Suppurative Lesions," Arch. Surg. **16**:312 (Jan.) 1928.

26. Yates (footnote 25, second reference).

27. Meyer, W. (footnote 25, second reference, p. 309).

28. Torek, F. (footnote 25, second reference, pp. 316-317).

29. Eggers, C., and Kernan, J. D.: Acute Pulmonary Suppuration: Selective Action of Artificial Pneumothorax in the Treatment of this Disease, Arch. Surg. **16**:279 (Jan.) 1928.

30. Churchill, E. D., and Agassiz, A.: A Method for Separating the Air Breathed by the Right and Left Lungs, together with the Effect of Pulmonary Circulatory Changes on this Divided Breathing, Am. J. Physiol. **76**:6, 1926.

control of breathing.[31] It was devised to separate completely the respirations of the two lungs and is shown in diagram in figure 1. When anesthesia was complete, the trachea was exposed through a midline incision in the neck and opened horizontally at a point about 2 inches (5 cm.) above the sternal notch. Two metal tubes, which were subsequently to divide the trachea into two separate

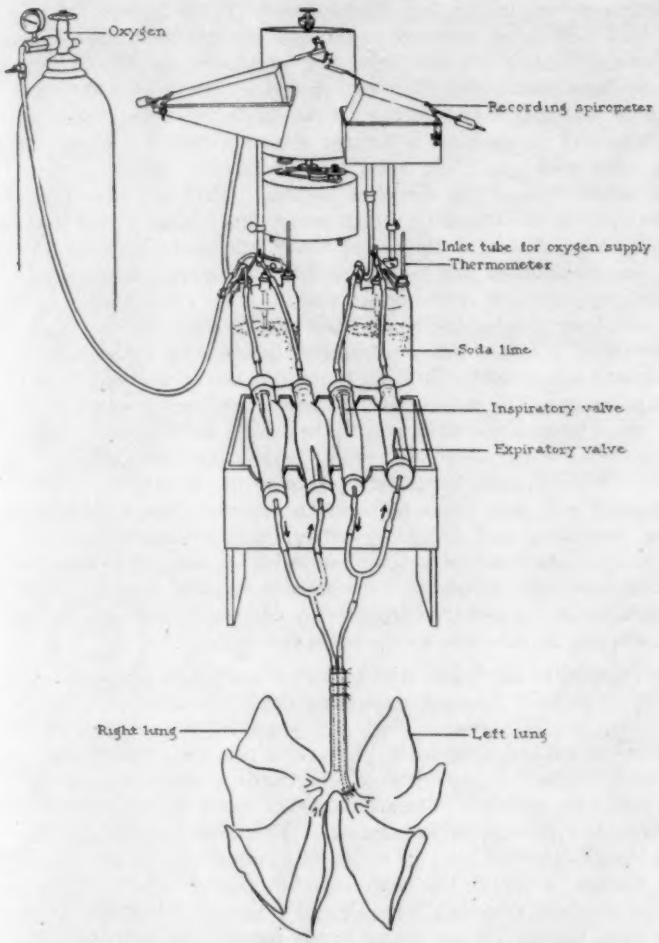


Fig. 1.—Diagrammatic view of the apparatus. The lower part of the sketch, which shows the arrangement of the tubes which were used for the separation of breathing, was copied from the diagram published by Churchill and Agassiz (Am. J. Physiol. **76**:6, 1926). The sketch shows the two separate respiration systems and the manner in which the respiratory curves were made.

compartments, were then inserted through the opening. The tubes were 19 and 21 cm. long and thin-walled, and both had an internal diameter of 7 mm. The

31. Moore, R. L.: A Study of the Hering-Breuer Reflex, J. Exper. Med. **46**:819, 1927.

longer one was curved at one end to conform with the angle at which the bronchus unites with the trachea. It had in addition two rubber rings at the extreme end with a narrow groove in between for receiving the ligature, which subsequently fastened it in the bronchus. This tube was passed downward into one bronchus. The shorter tube was straight. It was passed down to the opposite bronchus, but did not enter it. When the tubes were in place they were temporarily anchored by a ligature around the trachea about 1½ inch (3.7 cm.) below the opening. The animal was then given artificial respiration through each tube separately and turned on the right or left side, depending on whether the left or right bronchus was to be cannulated. The chest was opened in the fourth intercostal space by an incision beginning 1 inch (2.5 cm.) lateral to the edge of the sternum and extending around the chest for a distance of approximately 3 inches (7.6 cm.) and the ribs were spread and held apart by a mechanical retractor. The lung was reflected anteriorly and the bronchus isolated. This was done almost entirely by blunt dissection to avoid cutting nerve fibers and reduce bleeding to a minimum. After placing a ligature of heavy silk behind the bronchus, the long cannula was manipulated into position and firmly ligated. Expansion of all lobes of the lung subsequent to this indicated that the correct position had been obtained. Before completely closing the thorax, the lungs were expanded to expel the air from the chest. Closure was accomplished by suturing the intercostal muscles. When closure was complete, artificial respiration was discontinued and the animal breathed naturally. The opening in the trachea was now closed by packing muscle into the space between the tubes and tracheal wall down to the "anchor" ligature and compressing the trachea and muscle against the tubes with two additional ligatures. This procedure completely separated the breathing of the two lungs. Each cannula was now connected with a separate closed respiration system, including inspiratory and expiratory valves, sodium lime for removing carbon dioxide and a small spirometer for recording oxygen absorption curves and changes in respiratory movement. All animals breathed from 90 to 95 per cent oxygen throughout the period of observation. As the oxygen was absorbed, it was replaced through an inlet tube as shown in the diagram.

Every experiment was begun with a series of control observations. The oxygen absorption, respiratory rate and respiratory depth (tidal air) of each lung were obtained from the kymographic tracings. Representative samples of respiratory tracings are reproduced in figure 2. The pulse rate was counted with the aid of a stopwatch. While the tracings were being made, samples of arterial and mixed venous blood were collected. The arterial blood was withdrawn from the femoral artery through a cannula or by puncture. The mixed venous blood was taken from the right side of the heart by means of a special cannula introduced into that chamber through a slit in the right external jugular vein.[32] The samples of blood were carefully protected from air and transferred immediately to oxalated sampling tubes beneath oil and placed in the icebox until analyzed. The oxygen content of the samples was estimated by the Van Slyke and Neill[33] manometric method and the amounts of oxygen expressed in per cent by volume. All of the analyses were made in duplicate, and results which did not check within 0.5 per cent by volume were discarded.

---

32. Stewart, H. J.: The Oxygen and Carbon Dioxide Contents of the Arterial and Mixed Venous Blood in Normal Intact Dogs, J. Biol. Chem. **62**:641, 1925.

33. Van Slyke, D. D., and Neill, J. M.: The Determination of Gases in Blood and Other Solutions by Vacuum Extraction and Manometric Measurement, J. Biol. Chem. **61**:523, 1924.

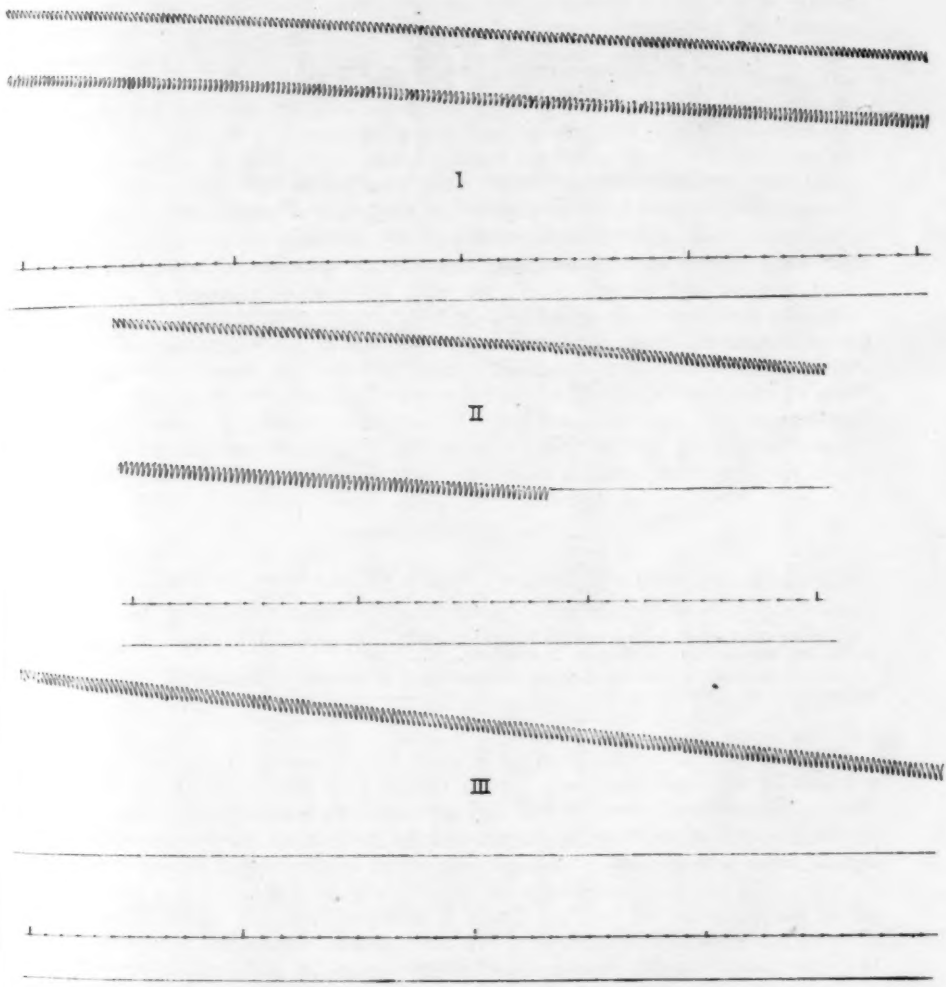


Fig. 2.—The respiratory tracings in experiment 7. The three sections were continuous and the upper and lower tracings were made by the right and left lungs respectively. In the upper tracing an excursion on the arc which is subtended by a chord 1 inch long represents a 231 cc. change in the volume of the recording spirometer. In the lower tracing, written by a smaller spirometer, an excursion on the arc which is subtended by a chord 1 inch long represents a volume change of only 141 cc. Section A shows the control period; section B, the point at which the left bronchus was blocked; and section C, the curve made by the right lung twenty minutes later. Time interval, five seconds.

The difference between the oxygen content of the arterial and of the mixed venous blood gives the amount of oxygen removed from the blood by the tissues or from the point of view of the lesser circulation the amount of oxygen taken up by the blood in passing through the lungs. With these one can calculate the total volume of blood flow per minute through the lungs according to the principle of Fick: [34]

$$\frac{\text{cc. of oxygen removed per minute}}{\text{amount of oxygen absorbed by 1 cc. of blood in passing through the lungs}} = \text{cc. of blood passing through the lungs per minute}$$

TABLE 1.—*Proportion of Oxygen Absorbed by Right and Left Lungs Respectively after Cannulation of One Main Bronchus and Complete Separation of the Breathing*

		Oxygen Absorbed	
Experiment Number	Bronchus Cannulated	By Right Lung, Per Cent	By left Lung, Per Cent
8	Left	56.2	43.8
7	Left	62.2	37.8
6	Left	60.6	39.4
20	Right	63.8	36.2
21	Right	64.5	35.5
2	Left	59.0	41.0
10	Right	60.6	39.4
11	Right	57.4	42.6
22	Left	57.1	42.9
14	Left	49.7	50.3
16	Left	56.7	43.3
17	Left	59.0	41.0
3	Left	60.4	39.6
1	Left	64.2	35.8
Average amount of oxygen absorbed by right lung in thirteen experiments (experiment 14 omitted)		60.1	
Average amount of oxygen absorbed by left lung in thirteen experiments (experiment 14 omitted)			39.9

If the amount of oxygen absorbed by each lung is measured separately, as it has been in my experiments, one can go further than this, and with the same formula estimate the volume of flow through each lung separately, provided that the oxygenation of the blood is the same in the two lungs. In the presence of normal lungs and adequate ventilation, one would hardly suspect this of being otherwise, still less so with an increased alveolar oxygen tension which is present during the inhalation of oxygen. There is additional evidence which indicates that this was true in my experiments. In every instance except one (experiment 14), the proportion of oxygen absorbed by the right and left lungs was approximately in the ratio of 3 : 2, a ratio which, as a result of observations made on fourteen different animals (table 1) I have come to regard as normal in dogs. Furthermore, in thirteen of the fourteen experiments, the oxygen content of the mixed arterial blood (femoral artery) was not found to vary to any appreciable extent from the oxygen content of the aerated blood (table 2). If the blood returning from one lung was incompletely oxygenated, this observation would not have been possible. The oxygen content of the aerated blood (oxygen

34. Fick, A.: Ueber die Messung des Blutquantums in der Herzventrikeln, Verhandl. d. phys.-med. Gesellsch. **2**:16, 1870.

capacity) was estimated after rotating the sample of blood in a flask for ten or fifteen minutes, during which time it was exposed to air at room temperature.

When these observations were completed, the airway to one lung was suddenly occluded by clamping the rubber tube connection between the cannula and respiration valves, and after a variable period of time all of the studies were repeated. The latter studies, however, were not made until the blocked lung was in a state of complete collapse, demonstrated in every instance by an autopsy which was performed immediately after the blood specimens were taken. The oxygen absorption of the patent lung now represents the total oxygen absorption, and one can calculate the total volume of blood flow from the formula already given. The blood flow through the patent lung is calculated likewise by assuming that the oxygen content of the arterial blood returning from it is maximal or equal to the oxygen content of the aerated blood. The truth of this assumption is scarcely questionable in the light of the increased ventilation of the patent lung (table 7) and the fact that its oxygen absorption at this time is increased over the control by the amount that was being absorbed by the opposite lung before its bronchus was occluded. The circulation through the collapsed lung is obtained by subtracting this amount from the amount that represents the total flow. In this way, the maximal amount of blood that could be in circulation through the collapsed lung was calculated. This method of calculating the blood flow through each lung separately was described by Dock and Harrison.[18] If it is assumed that the venous blood is not completely oxygenated in the aerated lung, the calculations would yield still larger values for the blood flow through the patent lung, and consequently smaller values for the flow in the collapsed lung.

EXPERIMENTAL WORK

The Relative Amounts of Blood Passing Through the Right and Left Lungs After Cannulation of One Main Bronchus and Complete Separation of the Breathing.—The amount of oxygen absorbed by each lung separately was measured in fourteen experiments, and the experimental data are presented in tables 1 and 2. It will be noted that with one exception (experiment 14) the proportion of oxygen absorbed by the right and left lungs was approximately in the ratio 3:2. Excluding experiment 14, the percentage of oxygen absorbed by the right lung varied between 56.2 and 64.5, and averaged 60.1, whereas that absorbed by the left varied between 35.5 and 43.8, and averaged 39.9. I believe that these figures represent the normal ratio in dogs, not only for the oxygen absorbed by the right and left lungs, respectively, but also for the relative amounts of pulmonary blood flow. Evidence in support of this has already been given (see Method). The actual amounts of blood, expressed in cubic centimeters per minute, are given in table 2.

It was conceivable that the oxygen absorption of the lung on the side on which operation was performed might be impaired by the operative procedure, either through some restriction of the movements of the thoracic wall or by some change resulting from the necessary manipulation of the lung. This possible traumatic factor was con-

TABLE 2.—*Effect of Collapsing One Lung on Volume of Blood Flow Through the Lungs*

Experimental Number and Sex *	Date	Weight, Kg.	Time, Minutes	Conditions	Oxygen Content			Oxygen Saturation		Arteriovenous Oxygen Difference		Oxygen Absorption			Volume of Blood Flow		
					Arterial Blood (Femoral Artery) Per Cent by Volume	Mixed Venous Blood (Right Side of Heart), Per Cent by Volume	Aerated Blood (Oxygen Capacity), Per Cent by Volume	Arterial, Per Cent	Mixed Venous, Per Cent	Total, Per Cent by Volume	Functioning Lung (After Collapse), Per Cent by Volume	Right Lung, Cc. per Minute	Left Lung, Cc. per Minute	Total, Cc. per Minute	Through Right Lung, Cc. per Minute	Through Left Lung, Cc. per Minute	Total, Cc. per Minute
8	1/ 4/29	11.6	3 : 20	Control	22.9	20.8	23.2	98.7	89.7	2.1	....	27.0	21.0	48.0	1286	1000	2286
♀			3 : 42	Left bronchus blocked													
			3 : 57	Left lung collapsed	22.5	18.4	25.0	90.0	73.6	4.1	6.6	63.5	0	63.5	962	587	1549
7	12/19/28	18.1	3 : 40	Control	17.5	15.8	18.5	94.6	85.4	1.7	....	58.0	35.2	93.2	3412	2070	5482
♀			3 : 52	Left bronchus blocked													
			4 : 12	Left lung collapsed	18.8	16.3	19.5	96.4	83.6	2.5	3.2	93.5	0	93.5	2922	818	3740
6	12/13/28	13.3	3 : 30	Control	19.5	14.4	19.7	99.0	73.1	5.1	....	47.3	30.8	78.1	927	604	1531
♀			3 : 49	Left bronchus blocked													
			4 : 17	Left lung collapsed	19.5	14.9	21.4	91.1	69.6	4.6	6.5	77.0	0	77.0	1185	489	1674
20	4/16/29	16.9	4 : 45	Control	20.5	18.3	21.5	95.3	85.0	2.2	....	41.1	23.3	64.4	1868	1059	2927
♀			4 : 55	Left bronchus blocked													
			5 : 55	Left lung collapsed	19.8	16.1	19.5	101.0	82.6	3.7	3.4	61.2	0	61.2	1800	0	1654
21	4/30/29	11.5	3 : 30	Control	21.0	19.0	20.9	100.0	91.0	2.0	....	24.9	13.7	38.6	1245	685	1930
♂			3 : 32	Left bronchus blocked													
			4 : 32	Left lung collapsed	20.1	15.8	20.9	96.1	75.6	4.3	5.1	34.9	0	34.9	684	128	812

2	11/ 1/28	13.0	3 : 30	Control	13.8	11.8	15.1	91.4	78.1	2.0	....	39.8	27.7	67.5	1990	1385	3375
♂			3 : 40	Left bronchus blocked													
			4 : 55	Left lung collapsed	15.7	10.4	15.7	100.0	66.2	5.3	5.3	63.3	0	63.3	1194	0	1194
10	1/16/29	12.6	3 : 55	Control	21.8	17.6	22.9	95.2	76.9	4.2	....	47.0	30.5	77.5	1119	726	1845
♀			4 : 07	Right bronchus blocked													
			4 : 22	Right lung collapsed	20.4	12.8	23.5	86.8	54.5	7.6	10.7	0	72.3	72.3	275	676	951
11	1/23/29	12.0	4 : 40	Control	17.4	15.4	17.0	102.0	90.6	2.0	....	34.0	25.3	59.3	1700	1260	2960
♀			4 : 52	Right bronchus blocked													
			5 : 07	Right lung collapsed	15.9	12.2	18.0	88.3	67.8	3.7	5.8	0	60.0	60.0	588	1034	1622
22	5/21/29	12.5	2 : 35	Control	16.1	13.1	16.0	100.0	81.9	3.0	....	28.8	21.6	50.4	960	720	1680
♂			2 : 47	Right bronchus blocked													
			3 : 02	Right lung collapsed	13.5	7.9	17.0	79.4	46.5	5.6	9.1	0	49.5	49.5	340	544	884
14	2/20/29	9.5	3 : 25	Control	21.2	17.4	21.6	98.1	80.6	3.8	....	34.5	35.0	69.5	908	921	1829
♂			3 : 37	Right bronchus blocked													
			4 : 37	Right lung collapsed	20.4	14.9	22.0	92.7	67.7	5.5	7.1	0	76.3	76.3	313	1074	1387
16	3/ 6/29	18.0	3 : 36	Control	19.4	16.6	18.9	102.6	87.8	2.8	....	46.6	35.6	82.2	1664	1271	2935
♂			3 : 40	Right bronchus blocked													
			4 : 40	Right lung collapsed	16.1	11.7	19.3	83.4	60.6	4.4	7.6	0	95.3	95.3	912	1254	2166
17	3/13/29	13.5	2 : 20	Control	22.0	19.6	22.3	98.7	87.8	2.4	....	36.4	25.4	61.8	1517	1058	2575
♀			2 : 29	Right bronchus blocked													
			3 : 35	Right lung collapsed	20.0	13.5	22.9	87.3	59.0	6.5	9.4	0	64.8	64.8	308	689	997
3	11/14/28	12.0	2 : 10	Control	20.0	19.0	20.1	99.5	94.5	1.0	....	27.5	18.0	45.5	2750	1800	4550
♂			3 : 27	Left bronchus blocked	Blood flow studies not possible because cannula for withdrawing mixed venous blood was incorrectly placed												
			5 : 00	Left lung collapsed	....	....	....	.....	....	...	....	....	....	51.9	....	....	....
1	10/26/28	18.3	5 : 25	Control	....	....	....	.....	....	...	....	53.0	29.5	82.5	....	....	....
♂					Blood flow studies not possible because cannula for withdrawing mixed venous blood was in the superior vena cava; experiment was not completed												

* In this table ♂ indicates male; ♀, female.

trolled by cannulating the left bronchus in some experiments and the right bronchus in others. The results were the same under both conditions.

The Changes in Pulmonary Circulation Following Collapse of the Left Lung.—The left bronchus was totally occluded and the left lung rendered completely airless in six experiments. Estimations of the effect

TABLE 3.—*Summary of Effect of Occluding Left Bronchus (Collapsing Left Lung) on Volume of Blood Flow Through Lungs* *

Experiment Number	Duration of Bronchial Occlusion, Minutes	Degree of Change in Volume of Blood Flow		
		Total, Per Cent	Right Lung, Per Cent	Left Lung, Per Cent
8	17	—31.0	—25.0	— 41.1
7	20	—32.0	—14.4	— 60.5
6	29	+ 9.3	+21.6	— 19.0
20	60	—43.5	— 3.6	—100.0
21	60	—57.9	—45.1	— 81.3
2	75	—64.6	—66.6	—100.0

* Increase is indicated by +; decrease by —. In every instance the "blocked" lung was completely collapsed (apneumatic) when these observations were made.

Macroscopic examinations were supplemented by microscopic examinations in experiments 6 and 7.

TABLE 4.—*Proportion of Blood Passing Through the Right and Left Lungs Before and After Collapse of the Left Lung*

Experiment Number	Volume of Blood Flow During Control Period		Volume of Blood Flow After Collapse of Left Lung	
	Right Lung, Per Cent	Left Lung, Per Cent	Right Lung, Per Cent	Left Lung, Per Cent
8	56.2	43.8	62.0	38.0
7	62.2	37.8	78.1	21.9
6	60.6	39.4	70.8	29.2
20	63.8	36.2	100.0	0
21	64.5	35.5	84.2	15.8
2	59.0	41.0	100.0	0
Average	61.1	38.9	82.5	17.5

on blood flow in the different animals were made at intervals which varied from seventeen to seventy-five minutes after the bronchus was occluded. In every instance the volume of blood per minute through the left lung showed a substantial decrease (from 19 to 100 per cent), which was most pronounced after the longer intervals. The average decrease in volume of blood per minute through the left lung was 67 per cent. In five experiments (8, 7, 20, 21 and 2), the cardiac output and the flow through the opposite (right) lung likewise diminished, but to a less extent. The latter observations showed a slight increase in

experiment 6. The proportion of blood passing through the right and left lungs during the control period averaged 61.1 and 38.9 per cent, respectively. After collapse of the left lung, the average percentage of blood passing through the right lung increased to 82.5, and that passing through the left decreased to 17.5. The actual values are shown in table 2 and the percentage changes in tables 3 and 4. In general, these results agree with those which were obtained by Loewy and von Schrötter,[4] Adams and Morris,[7] Andrus[9] and Coryllos and Birbaum.[10] It is worthy of note that both Hess[5] and Le Blanc,[8] who concluded that the pulmonary circulation in a blocked lung was not decreased, noted at autopsy that the lungs of their animals were inflated.

The Changes in Pulmonary Circulation After Collapse of the Right Lung.—These experiments were similar in every respect to those

TABLE 5.—*Summary of Effect of Occluding Right Bronchus (Collapsing Right Lung) on Volume of Blood Flow Through the Lungs* *

Experiment Number	Duration of Bronchial Occlusion, Minutes	Degree of Change in Volume of Blood Flow		
		Total, Per Cent	Right Lung, Per Cent	Left Lung, Per Cent
10	15	—48.4	—75.5	— 6.9
11	15	—45.2	—64.8	—17.9
22	15	—47.4	—64.6	—24.4
14	60	—24.2	—65.5	—14.2
16	60	—26.2	—45.0	— 1.3
17	66	—61.0	—80.0	—35.0

* Decrease is indicated by —. In every instance the "blocked" lung was completely collapsed (apneumatic) when these observations were made.

Macroscopic examinations were supplemented by microscopic examinations in experiments 10, 14 and 16.

reported under "Changes in Pulmonary Circulation Following Collapse of the Left Lung," with the one exception that the right lung was collapsed instead of the left. The intervals of time allowed to elapse between bronchial occlusion and the second estimations of the volume of blood flow varied in this group from fifteen to sixty-six minutes. Just as in the former series, all animals showed a conspicuous decrease in the volume of blood flow per minute through the collapsed lung. The decrease varied from 45 to 80 per cent, averaging 65.9, just a trifle less than the corresponding figure for the left lung. All animals likewise showed a noticeable decrease in cardiac output and a similar, though less striking, change in the volume of flow through the functioning lung.

The change in the proportion of blood passing through the two lungs was equally as striking. In these six experiments during the control period, the percentage of the cardiac output that passed through the right and left lungs averaged 56.8 and 43.2, respectively. After

collapse of the right lung, these values were reversed to 33.2 for the right lung and 66.8 for the left. The experimental data are presented in tables 2, 5 and 6.

The Effect of Total Collapse of One Lung on the Ventilation of the Opposite Lung and on the Respiratory and Pulse Rates.—These data are shown in table 7. As we expected, the depth of breathing of the opposite lung was consistently increased and the degree of increase was most marked after right-sided collapse. The effect on respiratory rate was variable. In three instances (experiments 7, 20 and 10), the rate showed practically no change. In one experiment (experiment 6) it decreased slightly, and in the other eight experiments it was regularly increased. The volume per minute of pulmonary ventilation of the

TABLE 6.—*Proportion of Blood Passing Through the Right and Left Lungs Before and After Collapse of the Right Lung*

Experiment Number	Volume of Blood Flow During Control Period		Volume of Blood Flow After Collapse of Right Lung	
	Right Lung, Per Cent	Left Lung, Per Cent	Right Lung, Per Cent	Left Lung, Per Cent
10	60.6	39.4	28.9	71.1
11	57.4	42.6	36.3	63.7
22	57.1	42.9	38.5	61.5
14	49.7	50.3	22.6	77.4
16	56.7	43.3	42.1	57.9
17	59.0	41.0	30.9	69.1
Average	56.8	43.2	33.2	66.8

breathing lung, obtained by multiplying tidal air (expansion) by respiratory rate per minute, was conspicuously increased in all of the experiments, illustrating the remarkable degree to which one lung can compensate when the function of the other is destroyed. Under the conditions in my experiments, one lung (either the right or left) could easily do the work of two.

Usually the pulse rate did not vary, or the variation was so slight that it was not significant. This indicates that the striking decrease in cardiac output per minute which followed collapse of one lung was accompanied by a proportional decrease in the stroke output of the heart. Therefore, it would appear that experimental collapse of one lung during the inhalation of oxygen relieved the heart of part of its burden.

Postmortem Examination.—After the final observations, each experiment was terminated by the intravenous injection of from 20 to 30 cc. of a saturated solution of magnesium sulphate, and all animals were subjected to immediate autopsy. The cannula to the nonblocked lung was occluded before the thorax was opened. This prevented partial deflation of the lung when the negative intrathoracic pressure was

destroyed and afforded an opportunity to compare the sizes of the two lungs under controlled conditions. Histologic examinations were made in five experiments. The sections were fixed in formaldehyde, embedded in paraffin and stained with eosin and methylene blue. The pathologic changes were the same in all instances. The lung, the bronchus of which had been occluded during life (collapsed lung), was found to be only about one-third the size of the breathing lung. It was deep red,

Fig. 3.—Photograph of the lungs from experiment 6. The left lung was airless, dark red, firm to palpation, heavy and sank in water. The right lung was expanded throughout the complete extent of all the lobes.

firm to palpation, heavy and rapidly sank in water. Its appearance resembled that of normal liver, and its cut surface freely exuded dark red blood. In striking contrast, the opposite lung was distended and of a normal pink appearance. The pleural cavity on the side of collapse regularly contained from 10 to 30 cc. of hemorrhagic fluid.

Sections of the collapsed lobes showed complete obliteration of the alveolar spaces. The alveolar septums were everywhere in intimate contact with each other, and the capillaries appeared to be congested

with red blood corpuscles. Numerous mononuclear leukocytes were present in all the sections, and small foci of blood pigment were frequently observed.

A photograph of the lungs from experiment 6 is reproduced in figure 3, and photomicrographs of sections of the lungs from experiment 6 are shown in figures 4, 5 and 6.

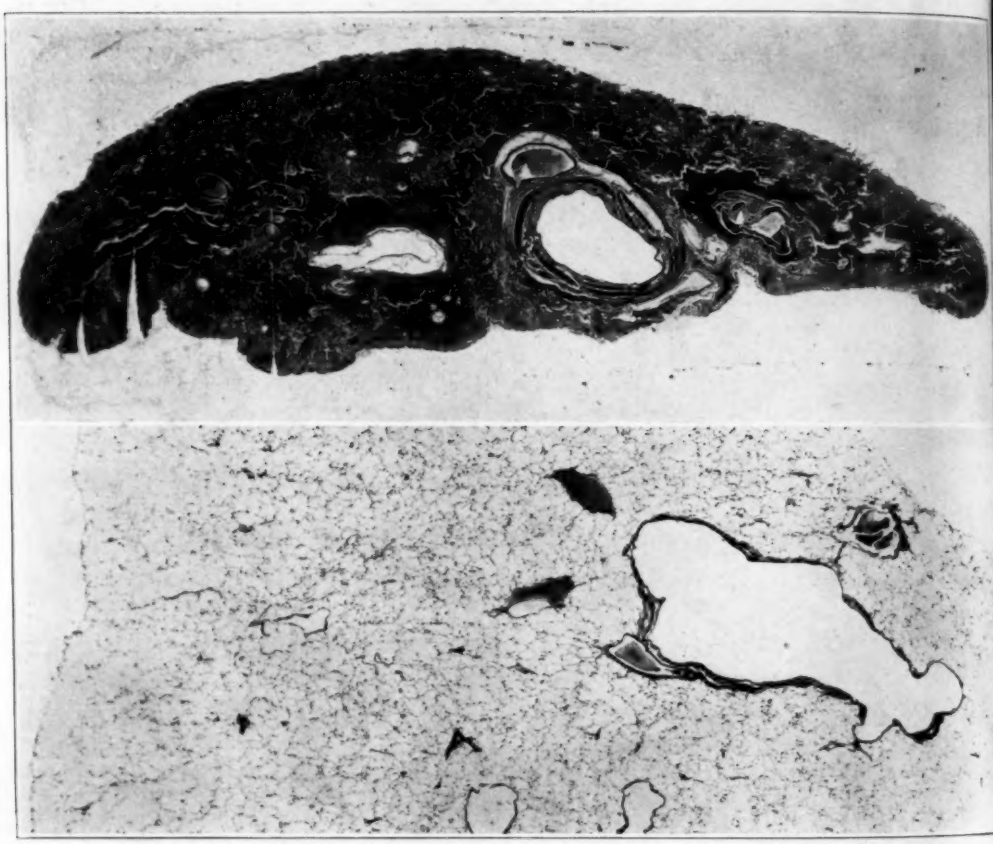


Fig. 4.—Photomicrograph of sections of the upper lobes of the lungs from experiment 6. Reduced from a magnification × 10. The upper picture shows complete obliteration of the alveolar spaces of the collapsed (left) lung. The right lobe (lower picture) is air-containing and presents a normal lung architecture.

The collapse of a lung which follows bronchial occlusion results from absorption of the gas that is trapped in the blocked alveoli. When the alveoli contain a high percentage of oxygen, this phenomenon occurs very rapidly, because the oxygen readily combines with the hemoglobin of the circulating blood. As the oxygen is removed, the alveolar walls

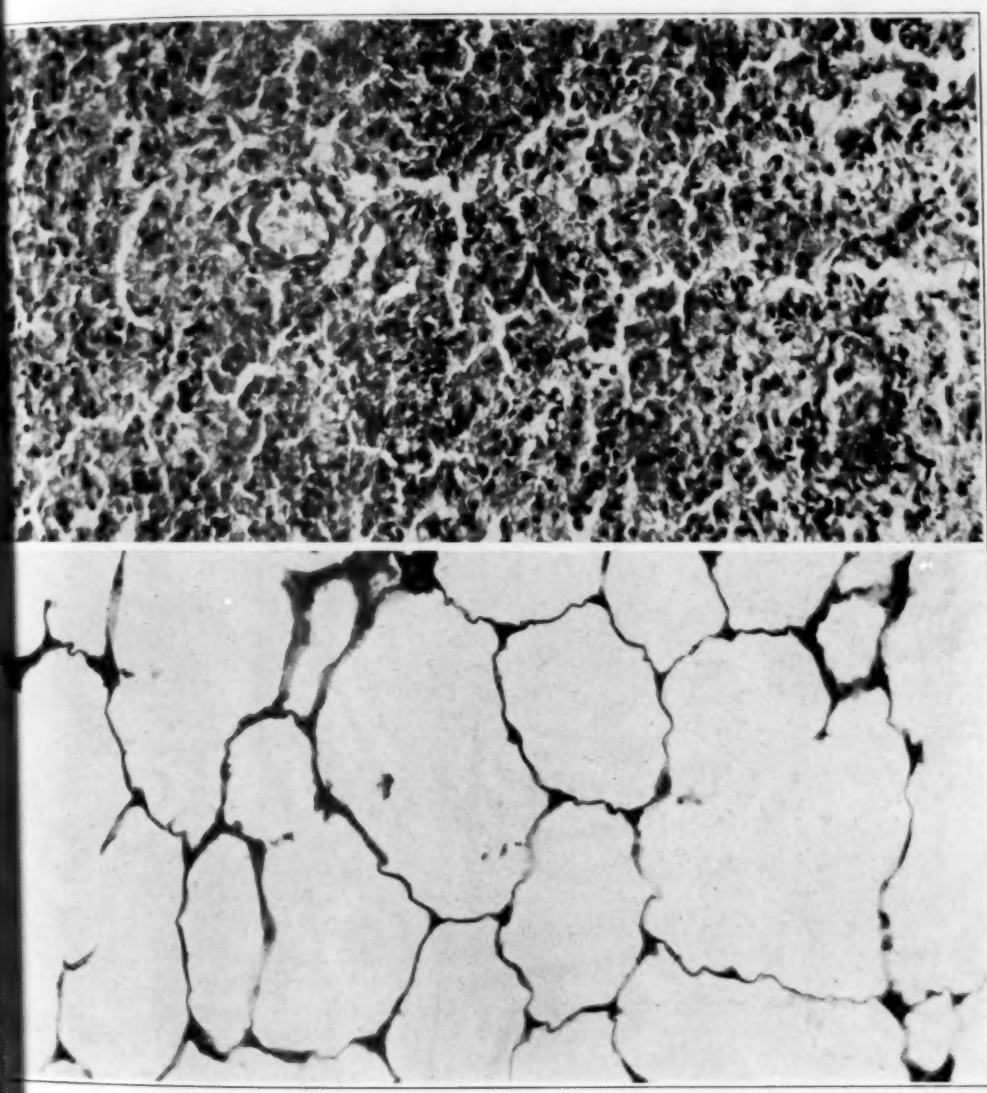


Fig. 5.—Photomicrograph of sections of the upper lobes of the lungs from experiment 6. Reduced m a magnification × 100. In the upper picture (left lung) the alveolar septums are in intimate con- with each other and the capillaries are tortuous and engorged with red blood cells. The black cks which resemble bacteria are fragments of blood pigment. The lower picture (right lung) does show any appreciable variation from normal.

shrink and fall together, and eventually the lung becomes airless. Deflation occurs much more slowly if a bronchus is occluded during the inhalation of air, because under these conditions the alveoli contain a large percentage of nitrogen with which gas the blood is normally saturated. These phenomena have been observed by Moore and Har-

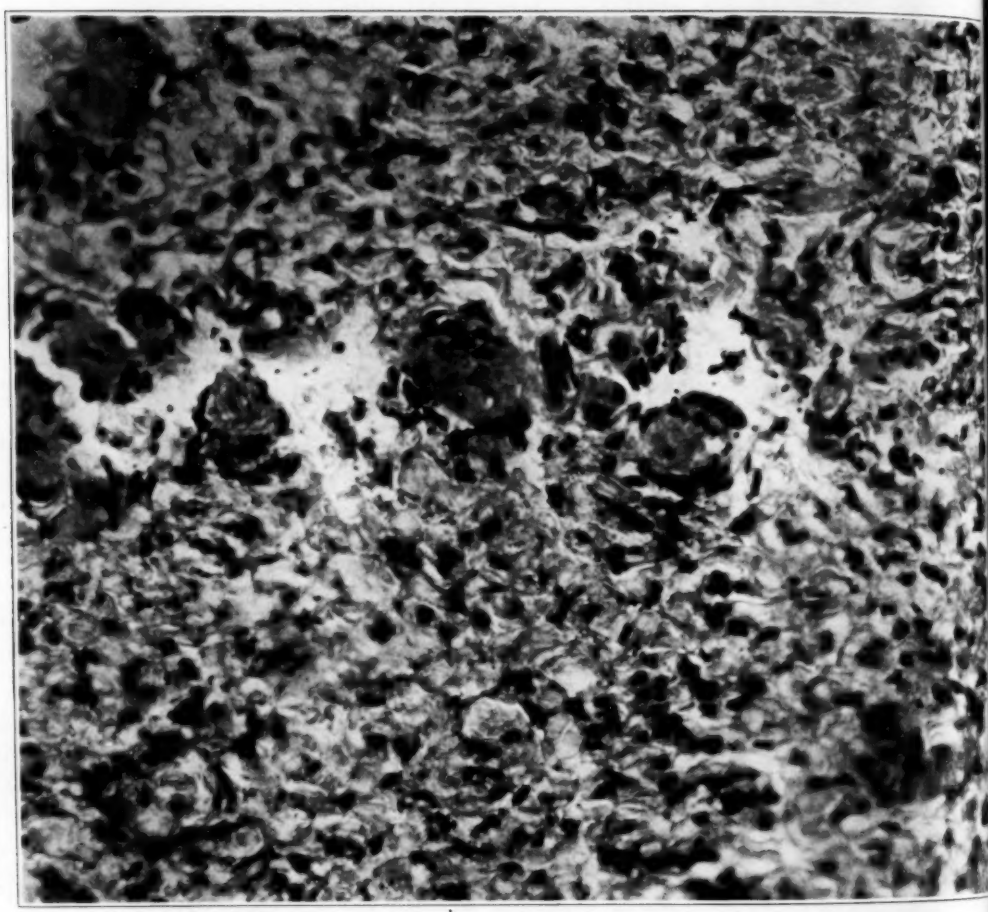


Fig. 6.—Photomicrograph of section of the left upper (collapsed) lobe from experiment 6. Reduced from a magnification × 400. Here the engorgement of the capillaries is shown in greater detail.

rison [35] when the chest was open and the animal was receiving artificial respiration. This mechanism for collapse was first described by Lichtheim [36] and was recently emphasized by Coryllos and Birnbaum.[1]

35. Moore, R. L., and Harrison, T. R.: Unpublished experiments.

36. Lichtheim, L.: Versuche über Lungenatelektase, Arch. f. exper. Path. u. Pharmakol. **10**:54, 1878.

COMMENT

The lesser circulation is concerned primarily, if not entirely, with the absorption of oxygen and the elimination of carbon dioxide and heat. This is rendered possible by the alternate expansion and collapse of the lungs (pulmonary ventilation). Through this mechanism air is brought into close contact with the circulating blood, the alveolar supply of oxygen is repeatedly replenished, and the elimination of carbon dioxide is accelerated. The amount of oxygen that will be removed from the lungs by the blood in a unit of time (provided the supply is adequate) will largely depend on the amount of reduced hemoglobin with which the oxygen comes in contact. I know from clinical experience, and my experiments have demonstrated clearly that one healthy lung is sufficient to take care of the oxygenation of the blood. This can be done with greater ease and with less strain on the circulation when the alveolar oxygen tension is increased by the administration of oxygen. If the ventilation of one lung is severely impaired by pneumothorax, bronchial occlusion or consolidation, the supply of oxygen to this lung may be diminished to the point of being inadequate. Under such conditions the blood that leaves this lung will enter the systemic circulation with a low oxygen content, and anoxemia will result. If, however, a diminution in blood flow accompanies the decrease in ventilation and not an increase in flow, as maintained by some authors, anoxemia may not follow, or its degree will be less. Therefore, from the physiologic standpoint it is easier to conceive of a diminished pulmonary ventilation, which is accompanied by an increased pulmonary blood flow, working to the disadvantage rather than to the advantage of the organism; whereas simultaneous and proportionate decrease in both of these functions, such as occurred in my experiments, would be advantageous.

Furthermore, that a beneficial effect on a healing process would come from an increased venous blood supply, as maintained by Cloetta [15] and Yates,[26] is open to question. In regard to the lung, it is not known to what extent venous blood shares the responsibility of nourishing lung tissue. That the bronchial supply is adequate for this is proved by the fact that necrosis of lung tissue does not follow ligation of the pulmonary artery.[37]

This paper would not be complete without a discussion of the possible causes of the diminished pulmonary circulation in the collapsed lung. In this condition, in addition to the actual decrease in the amount of blood passing through the collapsed lung in a unit of time, two other

37. Schlaepfer, K.: The Effect of the Ligation of the Pulmonary Artery of One Lung without and with Resection of the Phrenic Nerve, Arch. Surg. **13**:623 (Nov.) 1926. Moore, R. L.: Unpublished experiments.

observations stand out in importance: (1) the relative decrease in the fraction of blood passing through the blocked lung (as compared with the relative increase in the fraction passing through the functioning lung) which occured after collapse, and (2) the accompanying decrease in cardiac output. The decrease in the proportion of blood passing through the collapsed lung indicates that local changes within the collapsed lung itself were at least partly responsible for the decrease in its circulation. Of the possible local factors, two are especially deserving of consideration: first absence of lung motion and, second, mechanical obstruction in the capillaries.

The Significance of Immobilization of the Lung.—In regard to this factor, it is frequently stated in the literature that the alternate expansion and collapse of the lungs supports the flow of blood through the capillaries. In my experiments, the elastic stretching and recoil of one entire lung was destroyed. What was the effect of this immobilization of the lung? Could it have led to stasis in the vascular bed, which in turn precipitated the circulatory changes that were observed?

In attempting to answer these questions it is at least of some significance to call attention to the fact that total immobilization of a lung does not completely interrupt the pulmonary blood flow. My results show this. Furthermore, if immobilization of a lung did completely interrupt the pulmonary blood flow, total collapse would not follow so quickly in the footsteps of bronchial occlusion. Total collapse of a blocked lung that contains oxygen presupposes absorption of the oxygen by the pulmonary blood stream. This is necessarily so because the oxygen tension within the alveolar spaces is always greater than the oxygen tension of the venous blood. The character of the oxygen absorption curves is still more convincing that complete collapse of a lung which comes on so soon after bronchial occlusion (when an animal is breathing oxygen) is due to absorption of the trapped oxygen by the venous blood. I have noted repeatedly in all of my experiments that for the first few minutes following bronchial occlusion the oxygen absorption of the opposite lung is either unchanged or only slightly increased. Then the curve becomes steeper, and the oxygen absorbed by this lung equals the amount that was being absorbed by both lungs when the control observations were made. The change does not come for several minutes, because it takes this long for the oxygen trapped in the blocked lung to be removed by the blood.

In another study,[38] I reproduced tracings showing that partial occlusion of one main bronchus of a dog was followed by a decrease in the amount of oxygen absorbed by the corresponding lung and an

38. Moore (footnote 31, fig. 2, p. 827).

increase in the amount absorbed by the opposite lung. Under the experimental conditions that existed, the decrease in the oxygen absorption of the partially occluded lung most probably represented a corresponding decrease in the pulmonary blood flow, because the lung was not deflated and the supply of oxygen was not impaired. This observation suggests that limitation, or absence, of lung motion may contribute to the reduced circulation in pulmonary collapse. The fact that the pulmonary circulation is not destroyed when a lung is immobilized, as brought out at the beginning of this paragraph, does not imply that it is not reduced.

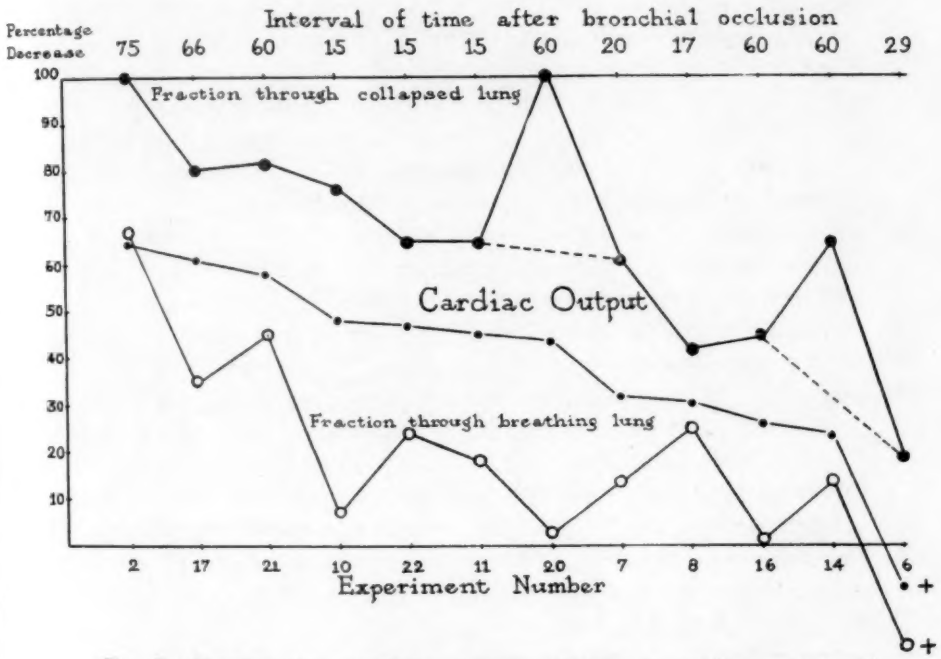


Fig. 7.—Showing the correlation between changes in cardiac output and fractions of blood passing through the collapsed and breathing lungs. Changes followed occlusion of one main bronchus and absorption of the gases trapped in the alveoli of the corresponding lung. The numbers at the top of the chart represent the intervals of time in minutes which were allowed to elapse between bronchial occlusion and these second estimations of blood flow. The numbers below represent the different experiments. A complete explanation of the chart is given in the text.

The Significance of Obstruction in the Capillary Bed.—Most of those writers who have described a diminished pulmonary circulation in a collapsed lung have attributed the decrease to a mechanical obstruction in the capillary bed. They have said that such an obstruction is caused

by the winding and coiling of the vessels which accompany contraction of lung tissue. I have not produced any evidence either for or against this opinion. My microscopic sections show tortuosity of the capillaries, but it would be purely theoretical to conclude from this that the flow of blood is retarded. One cannot estimate function from morphologic appearances.

The Significance of the Decrease in Cardiac Output.—If local changes within the lung itself were wholly responsible for the decreased flow of blood through the collapsed lung, one would expect some increase in the circulation through the functioning lung. This occurred, however, only once in my experiments (experiment 6). In eleven instances of twelve the reduced flow through the collapsed lung was accompanied by a decrease not only in the volume of flow through the breathing lung, but also in the total cardiac output. These changes have been correlated in figure 7, and a study of the curves sheds an interesting light on the interpretation of the circulatory adjustments. The changes in cardiac output (expressed in percentage of the initial values) in all experiments are arranged in decreasing order of magnitude, and against each measurement are plotted the simultaneous changes in the fractions of blood passing through the right and left lungs. If experiments 20 and 14 are overlooked, it will be observed that the decrease in circulation through the collapsed lung closely parallels the decrease in cardiac output. It is reasonable to assume that the decrease in cardiac output was precipitated by bronchial occlusion, or one of the consequences of bronchial occlusion (such as immobilization, or collapse of the lung), because the former always followed the latter. This conspicuous decrease in cardiac output is interesting, because it accounts for the rather striking percentage decreases in the fractions of blood passing through both the deflated and the breathing lungs, and, furthermore, because it offers an explanation of the marked differences that were observed in the percentages representing reduced flow in the collapsed lungs. When the left lung was collapsed, the percentage of decrease was greater after the longer intervals, but the time element did not offer a satisfactory explanation for all of the observations. The close parallelism, however, observed between the decrease in circulatory flow through the collapsed lung and the decrease in cardiac output, indicates plainly that the two were related to each other. To what extent, however, the circulation through the collapsed lung was affected by local changes within the lung itself and to what extent by the accompanying decrease in cardiac output, one is not able to say.

The curve that represents the fractions of blood passing through the breathing lungs is more irregular. Theoretically, the irregularity of this

curve could be caused by varying fractions of blood which were shunted through the breathing lung, either by absence of lung motion or obstruction in the vascular bed on the side of collapse.

Just why the decrease in cardiac output was moderate in some animals (it increased in one) and large in others is not clear. Nor is it completely clear why the cardiac output decreased at all. It is conceivable that stagnation of blood in the blocked lung was responsible for a part of the decrease. The increase in cardiac output that occurred in experiment

TABLE 7.—*The Effect of Occluding One Main Bronchus (Collapsing One Lung) on the Respiratory Rate, Pulse Rate and Expansion of the Opposite Lung**

Experiment Number	Conditions	Respiratory Rate per Minute	Pulse Rate per Minute	Tidal Air		
				Right Lung, Cc.	Left Lung, Cc.	†
8	Control	43	196	33	23	.....
	Left lung collapsed	50	204	55	0	66.6
7	Control	43	184	39	29	.....
	Left lung collapsed	44	...	53	0	35.9
6	Control	80	156	27	24	.....
	Left lung collapsed	77	154	40	0	32.5
20	Control	13	160	88	17	.....
	Left lung-collapsed	12	150	115	0	30.6
21	Control	23	108	41	25	.....
	Left lung collapsed	19	116	61	0	48.8
2	Control	21	148	35	35	.....
	Left lung collapsed	27	147	60	0	71.4
					Average	47.6
10	Control	13	175	52	96	.....
	Right lung collapsed	12	173	0	162	68.7
11	Control	20	160	57	27	.....
	Right lung collapsed	40	160	0	64	137.0
22	Control	31	162	41	37	.....
	Right lung collapsed	48	156	0	53	43.2
14	Control	30	120	32	27	.....
	Right lung collapsed	47	139	0	44	62.9
16	Control	19	165	55	43	.....
	Right lung collapsed	26	154	0	70	62.7
17	Control	41	128	36	34	.....
	Right lung collapsed	50	159	0	57	67.6
					Average	73.6

* For the interval of time elapsing between bronchial occlusion and the second observations see table 1.

† The figures in this column represent the percentage increase in the tidal air of the "opposite" lung.

6, however, makes this interpretation doubtful. It is equally difficult to connect the decrease in cardiac output with intrathoracic pressure changes. If it is assumed that the interpleural partition (mediastinum) in the dog is a freely movable structure and can shift over far enough toward the collapsed lung to equalize the pressure in the two pleural cavities, the amount of negative intrapleural pressure that is developed during inspiration after collapse probably does not differ appreciably from the normal. I believe that this was the situation in all of my experiments. Theoretically (from the standpoint of pressure changes alone), one would not have expected any variation in the filling or output of the heart.

One must exercise caution when applying the results of animal experimentation to clinical conditions. I realize this, but the urge to do research, with some at least, originates in the clinic, and the real pleasure comes when one applies one's ideas to the understanding and treatment of human disease. I undertook this study primarily because I was interested in the physiologic alterations that accompany collapse. Some of those investigators who have preceded me, however, notably Cloetta and Yates, entered into a theoretical discussion of the therapeutic value of the circulatory changes that accompany this condition. Both of these authors expressed the belief that when a lung is put in the position of incomplete deflation its pulmonary circulation is increased, and that this increase in circulation is one of the desired and valuable effects of artificial collapse in clinical conditions. I have not studied the circulation in a partially deflated lung, but my results suggest that in cases of complete deflation (apneumatosis) of a lung the changes in the pulmonary blood circulation would probably have a retarding influence on a diseased process because the blood flow is so strikingly reduced. I am inclined to believe that resting the lung is of greater therapeutic value than the accompanying circulatory changes.

Furthermore, I have produced experimentally a condition that is seen in a surgical ward most frequently as a postoperative complication, and it is now definitely known that many cases of clinical collapse are due to bronchial occlusion. This condition has received much comment in the literature within recent years. It is most often one-sided, and although a one-sided massive collapse is not fatal unless complications develop, the sufferers may be very uncomfortable, and are frequently dyspneic and anoxemic. That their anoxemia is not due entirely to the passage of fractions of blood through completely unaerated channels is suggested when the administration of oxygen relieves their cyanosis and allows them to breathe easier. When one lung of a dog is rendered functionless during the inhalation of oxygen, the reduction in the saturation of the arterial blood is usually slight. To me, this is an experimental confirmation of the advisability of giving oxygen in cyanotic cases with massive collapse.

SUMMARY

The respiratory movements and oxygen absorption curves of the right and left lungs were recorded separately in fourteen experiments on anesthetized dogs during the inhalation of oxygen. After total occlusion of one primary bronchus and complete collapse of the corresponding lung, similar tracings were taken of the opposite nonobstructed lung. Simultaneous estimations of the oxygen contents of the mixed venous,

arterial and aerated blood specimens were made. The data obtained have been used to calculate cardiac output and fractions of blood passing through the pulmonary system of each lung separately: first, under relatively normal conditions and secondly, at varying intervals after bronchial occlusion and complete collapse of either lung. It was found that:

1. When breathing was free, the amounts of oxygen absorbed by the right and left lungs, respectively, were approximately in the ratio of 3:2.

2. Under similar conditions, the fractions of blood that passed through the right and left lungs were in the same ratio.

3. Total occlusion of one primary bronchus was regularly followed by complete collapse of the corresponding lung.

4. Complete collapse of one lung (either the right or left) was accompanied by a conspicuous decrease in the pulmonary blood circulation on the affected side.

5. The cardiac output and the circulation through the opposite lung decreased to a less extent in eleven of twelve instances.

6. The percentage decrease of blood flow through the collapsed lung varied directly with the decrease in cardiac output.

CONCLUSIONS

The reduced pulmonary blood flow in a lung that has been completely collapsed by bronchial occlusion results in part from local changes within the lung itself and in part from the accompanying decrease in cardiac output. Of the local conditions, absence of lung motion is probably of some importance.

MULTIPLE NEUROFIBROMATOSIS (von RECKLINGHAUSEN'S DISEASE)

WITH SPECIAL REFERENCE TO MALIGNANT TRANSFORMATION *

KIYOSHI HOSOI, M.D.
Senior Eugene Littauer Research Fellow in Pathology
ALBANY, N. Y.

Since the epochal work of von Recklinghausen [1] in 1882, there has been much discussion about the origin and nature of neurofibromatosis. Although the literature contains hundreds of reported cases of multiple neurofibromatosis, the condition is not of frequent occurrence in the experience of any one physician. Malignant transformation of these cutaneous or deep-seated nodules is of unusual interest, especially when one is able to follow a case like the one here reported from the stage of benign neurofibroma to that of spindle cell sarcoma.

REPORT OF CASES

CASE 1.—*History.*—W. F., a white man, aged 28, was admitted in 1927 to the service of Dr. George E. Beilby, Albany Hospital, with the chief complaint of soreness in the right side of his neck and lumps on both sides of his neck. Except for the usual diseases of childhood, his general health had been excellent. In 1920, his tonsils and adenoids were removed. His family history was not remarkable. As regards his present illness, the patient knew he had had a "goiter" for about ten years, but it had been symptomless until July, 1927. At that time, he noticed a "drawing" and an ache in the side of his neck. The swelling itself did not increase in size; possibly it had grown slightly smaller. He had had headaches at night while at work in the railroad yards. There were no tremors of his hands, no sweating or flushing and no loss in weight.

Examination.—Physical examination showed a well developed and well nourished young man. There was no motor restlessness. The skin was soft and cool, and showed no flushing. There were numerous soft tumors in the skin, varying in size from about 2 mm. to 5 cm. in diameter. Most of these cutaneous nodules were sessile; others, pedunculated. Their distribution was irregular. The larger soft masses were on the right shoulder, the upper part of the left arm and the left knee. There was also an irregular brownish pigmentation of the skin. A few pigmented areas measured from 2 to 5 cm. in diameter, but most of them resembled freckles (fig. 1).

The skull was symmetrical. There was a slight tenderness over the right mastoid and some fulness on the right side of the pharynx as if a swelling on the outside were pressing inward.

The neck was large and asymmetrical, due to bilateral, large bosselated masses. The mass on the right side was the larger, and extended almost to the

* Submitted for publication, May 2, 1930.

* From the Department of Pathology, Albany Medical College, and the Pathological Laboratory of the Albany Hospital.

1. von Recklinghausen, F.: Ueber die multiplen Fibrome der Haut und ihre Beziehung zu den multiplen Neuromen, Berlin, A. Hirschwald, 1882, pp. 138.

tip of the mastoid process, beyond the midline and down behind the clavicle. The lower pole was not palpable. Pressure on the lower edge caused coughing. The mass on the left side was not so large; its lower pole was also not felt. The trachea was markedly displaced to the left. Many small, indurated discrete nodules were palpable on both sides of the neck. The total tumor mass was firm and somewhat irregular. A loud systolic bruit could be heard over it. There were large dilated veins over the right side of the neck and shoulder.

Fig. 1 (case 1).—Patient's back, showing the cutaneous nodules and the patchy pigmentation, appearing mostly as freckles.

The chest was well developed. The right supraclavicular fossa was almost obliterated, the left clavicle being the more prominent. There was a slight increased convexity of the right side of the chest posteriorly. The expansion of the chest was good and symmetrical. The lungs were everywhere resonant, except for a slight impairment at the apex of the right lung. The breath sounds were loud and bronchovesicular, especially at the apex and on the anterior and

posterior part of the right lung. The heart was not remarkable. The abdomen showed no important findings.

There were many bilateral, enlarged, indurated nodules in the cervical, axillary, epitrochlear and inguinal regions.

Roentgen examination by Dr. William P. Howard showed a large mass in the mediastinum extending above the heart into the root of the neck, which suggested a retrosternal thyroid gland, measuring 14 cm. in its greatest transverse diameter. The trachea was displaced toward the left and apparently compressed (fig. 2).

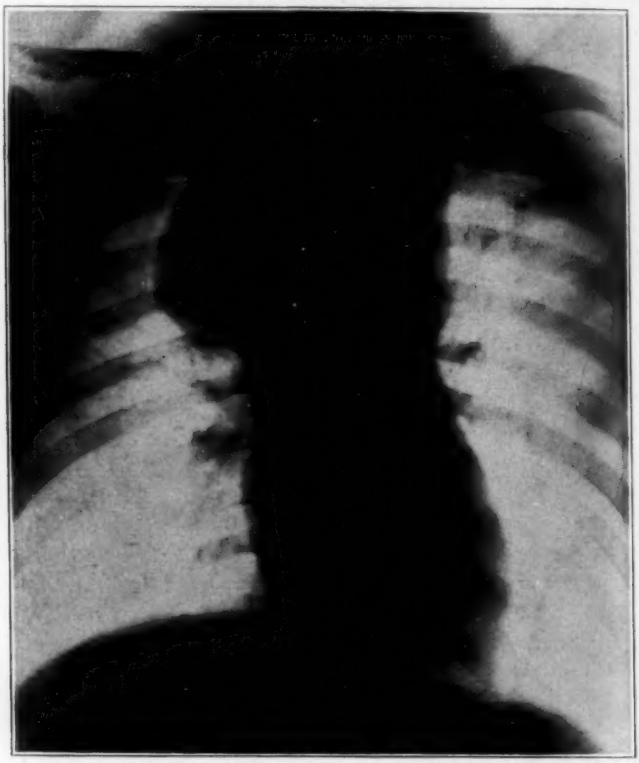


Fig. 2 (case 1).—Roentgenogram showing a very large mediastinal mass protruding into the right pleural cavity and extending from above the heart into the root of the neck. The trachea is displaced toward the left.

The urinalysis was of no interest. The Wassermann test of the blood was negative.

Operation.—At operation, an exploratory thyroid incision was made. The tumor mass extended behind the manubrium sterni and was soft. As total removal would necessitate resection of ribs, only a piece of the tumor was removed for diagnosis. The pathologic diagnosis by Dr. Victor C. Jacobsen was edematous fibroma. The patient was discharged improved.

Second Admission.—The patient was readmitted to the hospital in January, 1928, for further observation. Except for the operative scar, the physical signs were essentially the same as those previously described.

In July, 1928, the right side of the neck became enormously swollen. It was incised, and some degenerating necrotic material was allowed to drain. Roentgen examination at that time showed a large mass projecting from the root of the neck into the right side of the chest, displacing the trachea toward the left side. This area was approximately 13 by 12 cm.

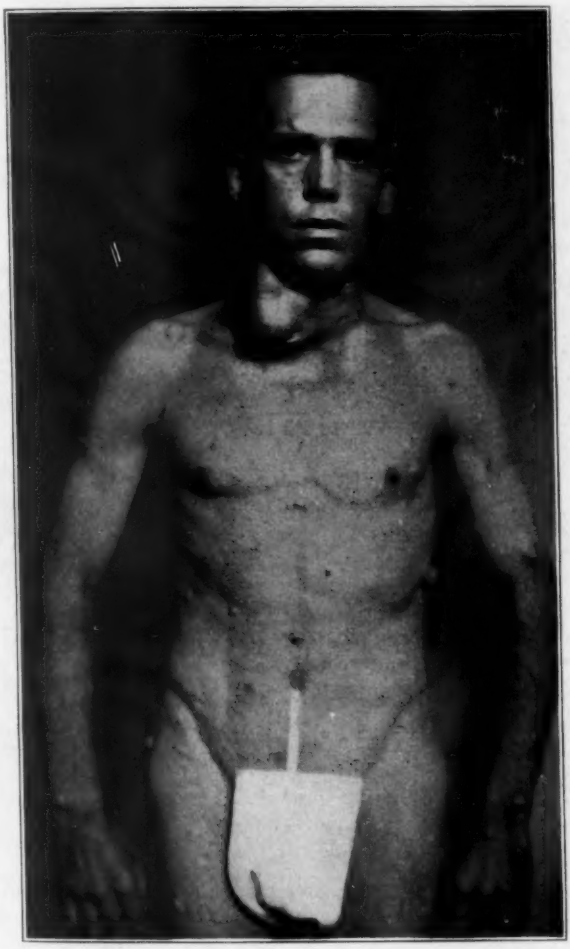


Fig. 3 (case 1).—Appearance of patient on his final admission. Note the large, irregular, almost pendulous tumor mass in the neck, the many small cutaneous fibromas, sessile and pedunculated, and the patches of pigmentation.

Third Admission.—The patient was admitted to the hospital for the third time in September, 1928. The chief complaints at this time were pain and ache in the left side of his head and in his left shoulder. Examination showed numerous sessile or pedunculated tumor masses in the skin along the course of different nerves, varying in size from 2 mm. to 5 cm. In the neck, there were large, asymmetrical, bilateral masses, larger on the right, which extended to the right

mastoid process (fig. 3). The lower pole extended behind the sternum. Pressure over this region caused coughing. The lower pole on the left side was also not palpable. The trachea was displaced to the left. The left clavicle appeared slightly prominent. Many other small discrete skin nodules were palpable.

At operation, the tumor masses were found to be markedly vascular. That portion projecting above the sternum was myxomatous and very friable. Anteriorly, it crossed the midline to the left, evidently invading muscle. Bleeding was controlled by sutures. Two drains were inserted in the cavity formed by the removal of the part of the tumor mass. The pathologist's diagnosis this time was spindle cell sarcoma.

After leaving the hospital, the patient continued to grow worse, and he died a month later. Autopsy could not be obtained.

Microscopic Examination of Tissue Removed in 1927.—The specimen was fixed in Zenker's solution. Paraffin sections were stained with hematoxylin and eosin, van Gieson's trinitrophenol fuchsin, Mallory's phosphotungstic acid

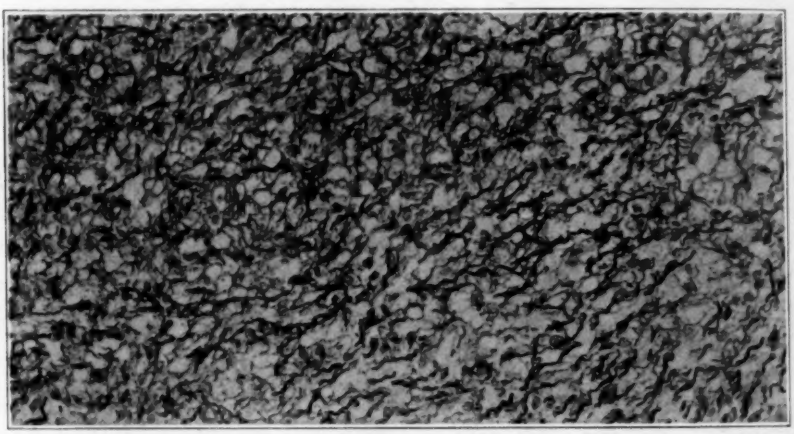


Fig. 4 (case 1).—Photomicrograph of a section of the large cervical tumor. The numerous reticulum fibrillae are in intimate association with the tumor cells. Foot and Mènard's silver stain, lightly counterstained with Harris' hematoxylin; × 197.

hematoxylin, Weigert's resorcin fuchsin, Foot and Mènard's ammonium silver and Laidlaw's lithium silver.

All the sections were very poor in cells, which were widely separated from one another by edematous stroma composed of little reticulum but of numerous thin, wavy strands of collagen. These cells were either spindle-celled or stellate in shape and small. The nucleus was generally ovoid, more or less vesicular, and had a minute insignificant nucleolus. There were no mitotic figures. No nerve trunks were demonstrable in the sections of the tumor proper.

Microscopic Examination of Tissue Removed in 1928.—These sections were very cellular. In fact, the cells were so densely packed together that hardly any collagen fibrils were demonstrable over wide areas. But when the silver stains were used, in intimate association with each tumor cell were delicate reticulum fibrillae which formed a fine intricate network (fig. 4). After the Laidlaw stain, the nuclei were silver-negative, so that only the fine dense meshwork

was seen. With the Mallory stain, fibroglia could be demonstrated streaming from the tumor cell. No elastic fibrils were present. Mitotic figures were very abundant. The tumor cells had invaded the striated muscles of the neck. The microscopic picture was now that of a rather anaplastic type of fibroblast, rapidly growing and infiltrating adjacent structures. The tumor cells were large, with conspicuous round or oval nuclei (fig. 5). Blood vessels of the capillary type were quite numerous. These tumor cells were often grouped more densely around these capillaries in a perithelial manner and had even invaded some vessels.

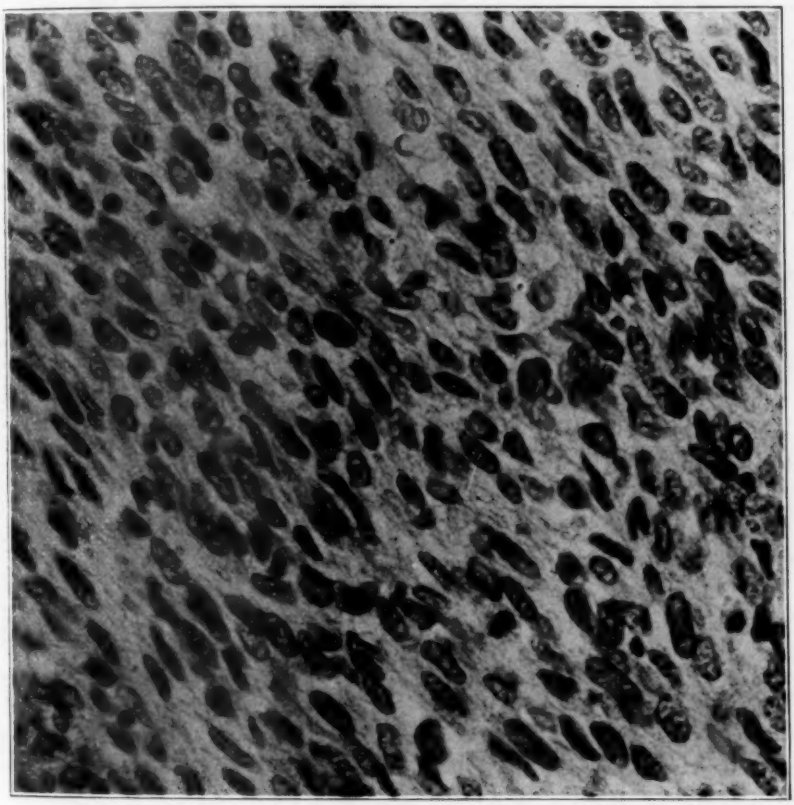


Fig. 5 (case 1).—Photomicrograph of a cellular area from the large cervical tumor. Note the three mitotic figures. Hematoxylin and eosin strain; × 248.

This invasion of blood vessels would indicate, although no necropsy could be obtained, that the patient probably died from internal metastasis as judged from the progressive cachexia just before his death. In some sections, there were large areas of intense edema, with cystic spaces containing coagulated fluid and strands of fibrin. Small hemorrhages were present here and there.

Fortunately, pieces of adjacent muscle tissues were also removed at the time of the last operation. In addition to the invasion of the muscle by the sarcoma as observed at operation, some interesting and important information was obtained on microscopic study. Serial sections were made of all the muscle blocks. Some

of the small nerves in the perimysium showed a more or less marked concentric proliferation of the perineurium (fig. 6); in others, the proliferating perineurium appeared like a crescent around the nerve.

As regards the muscle fibers themselves, most of them appeared normal. Scattered here and there were many typical muscle spindles. In addition, small collections of lymphocytes were present among the muscle fibers.

In some areas adjacent to the sarcoma, the perivascular tissues and even the walls of the arterioles and venules were infiltrated with polymorphonuclear leukocytes without, however, any thrombosis. In other areas, there was a dense perivascular lymphocytic infiltration. In the cystic portions of the tumor proper, a few of the venules showed hyaline mural thrombi.

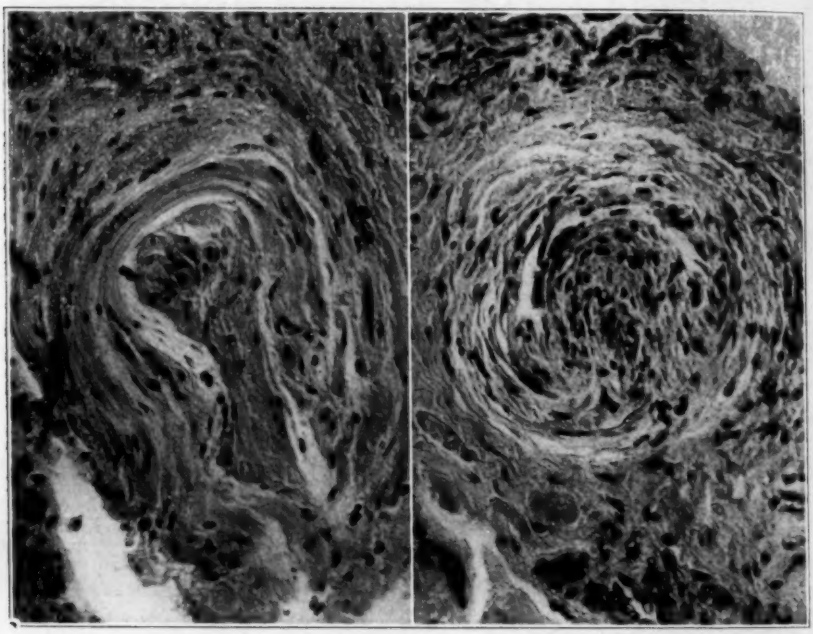


Fig. 6 (case 1).—Photomicrograph of two different small nerve bundles, showing the concentric proliferation of the perineurium. Mallory's eosin-methylene blue stain; × 306.

Case 2.—*History.*—F. L., a white man, aged 68, was admitted in 1929 to the service of Dr. Thomas Ordway, Albany Hospital, because of a troublesome, productive cough of two weeks' duration. The day prior to admission, he felt chilly and had a severe headache. He said that he had not been sick before. Several years ago, he fractured his right shoulder and several ribs; ever since then, the right side had hurt him whenever he coughed. A hernia had been present since childhood. He had been having nocturia (once) for the last several years. When he was 10 years old, small lumps began to develop in his skin. They had progressed in size and number up to the present time, but had never caused any inconvenience. His family history, including that of his five children, was entirely uninteresting, except for one important fact: one sister had small lumps over her body similar to his own condition.

Examination.—Physical examination of the nose, throat and chest showed nothing remarkable. No masses were felt in the abdomen. There was a large indirect inguinal hernia. Covering the entire trunk, so thickly scattered that it was impossible to find an area large enough to lay a stethoscope flat, there were soft nodules varying in size from that of a pinhead to that of a walnut. The larger nodules were pedunculated, soft and flabby, not tender, and covered with

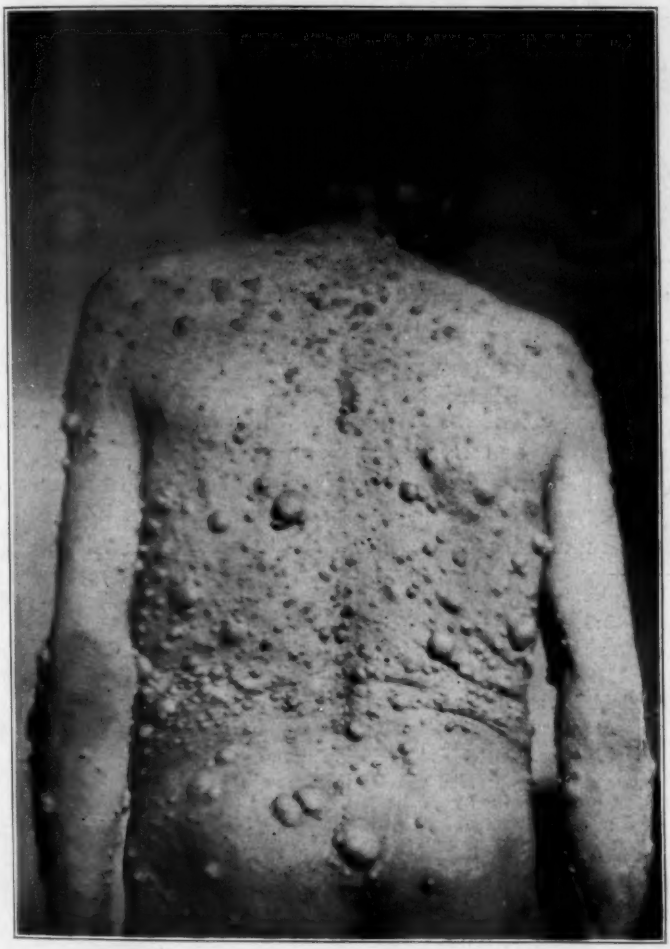


Fig. 7 (case 2).—Patient's back, showing the thickly set distribution of the mollusca fibrosa, varying in size from that of a pinhead to that of a walnut.

apparently normal skin (fig. 7). Some of these nodules were located deeply in the subcutis. They were present in great numbers on the various surfaces of all four extremities and on the neck, face, scalp and even the eyelids. They were most numerous on the trunk.

The urine was alkaline, showed a trace of albumin, no sugar and contained much cellular débris. The Wassermann and Kahn tests of the blood were negative. Chemical examination of the blood gave a nonprotein nitrogen of

36.6 mg. and a sugar of 90 mg. per hundred cubic centimeters. The hemoglobin was 90 per cent. The systolic blood pressure was 115 mm., the diastolic 80 mm.

None of the cutaneous nodules was excised for microscopic examination.

COMMENT

The mesodermal origin of multiple neurofibromas from the epineurium, perineurium and endoneurium of the nerve bundles is now generally accepted. However, Verocay [2] expressed the belief that they are ectodermal, arising from the cells of the sheath of Schwann. The adherents of the latter theory have given various names to these tumor formations—neurinoma (Verocay), schwannoma (modern French writers) and glioma (Bertrand and Charrier [3] and others). Whether the cells of the sheath of Schwann are specialized glia cells or not is still a much disputed point. According to Penfield,[4] multiple neurofibromatosis is a generalized fibrous connective tissue reaction showing both nerve fibers and connective tissue elements in the tumors. He classified the neurinoma, solitary neurofibroma, gliome périphérique and acoustic neuroma under the general heading of perineurial fibroblastoma. For a complete discussion of the subject, the reader is referred to the classic monographs of von Recklinghausen, Thomson,[5] Adrian,[6] Verocay and Herxheimer and Roth.[7]

Isolated primary fibrosarcomas and spindle cell sarcomas of the different nerves without an associated von Recklinghausen's disease have been reported by Hume,[8] Kriege,[9] Peet,[10] McArthur,[11] Villegas

---

2. Verocay, J.: Zur Kenntnis der "Neurofibrome," Beitr. z. path. Anat. u. z. allg. Path. **48**:1, 1910.

3. Bertrand, I., and Charrier, J.: Cystic Glioma of the Ulnar Nerve, Rev. neurol. **38**:1345, 1922.

4. Penfield, W.: The Encapsulated Tumors of the Nervous System, Meningeal Fibroblastomata, Perineurial Fibroblastomata and Neurofibromata of von Recklinghausen, Surg. Gynec. Obst. **45**:178, 1927.

5. Thomson, A.: On Neuroma and Neuro-Fibromatosis, Edinburgh, Turnbull & Spears, 1900, pp. 168.

6. Adrian, C.: Ueber Neurofibromatose und ihre Komplikationen, Beitr. z. klin. Chir. **31**:1, 1901.

7. Herxheimer, G., and Roth, W.: Zum Studium der Recklinghausen'schen Neurofibromatose, Beitr. z. path. Anat. u. z. allg. Path. **58**:319, 1914.

8. Hume, G. H.: A Case of Sarcoma of the Internal Popliteal Nerve, Lancet **2**:344, 1886; Cases of Tumor of Nerve Trunks, Illustrating the Results of Excision of the Tumor Along with the Affected Portion of Nerve, Lancet **2**:654, 1891.

9. Kriege, H.: Ueber das Verhalten der Nervenfasern in den multiplen Fibromen der Haut und in den Neuromen, Virchows Arch. f. path. Anat. **108**: 466, 1887.

10. Peet, M. M.: Spindle Cell Sarcoma Arising in a Cavernous Lymph- and Hem-angioma of the Musculospiral Nerve, J. Michigan M. Soc. **16**:320, 1917.

11. McArthur, L. L.: Sarcoma of the Posterior Tibial Nerve: Excision; Removal of Metastatic Foci in Retroperitoneal Lymph Glands Three Months Later, S. Clin. North America **4**:131, 1920.

and Rojas,[12] McGuire and Burden[13] and many others. The nerves involved in the foregoing cases were the sciatic, median, musculospiral, internal popliteal and posterior tibial. In Meyer's case,[14] the fibrosarcoma was of the hour-glass type with intradural and extradural portions extending from the sixth cervical to the first dorsal segments. Kredel and Beneke[15] described a spindle cell sarcoma, involving both the median and ulnar nerves, in an infant 2 months old. In Buford and Davis' case,[16] the tumor of the sciatic nerve at the first removal was a typical fibroma which at a later date had become sarcomatous. In 1900, Thomson collected twelve cases of authenticated primary sarcomas of nerves, 50 per cent of these occurring in the great sciatic nerve. Allenbach,[17] in 1921, collected twenty-four cases of sarcomas of the sciatic nerve, but added that in most only the gross description was given without microscopic observations. Garrè[18] differentiated between the primary sarcoma of nerves not associated with von Recklinghausen's disease and the secondary sarcoma of nerves in which the latter is due to a sarcomatous transformation of one of the preexisting neurofibromas. The primary form is similar to other types of sarcomas, being characterized by great malignancy. The growth reaches considerable size in a few months, invades the surrounding tissue and leads not infrequently without local or regional recurrence to internal metastasis. The patient succumbs to the generalized sarcomatosis in a short time. In great contrast to the foregoing form, in the secondary form one of the neurofibromas undergoes malignant transformation, frequently following trauma or often without known cause, and begins to grow rapidly. However, this growing tumor remains enclosed in its connective tissue capsule for a long time. Operative intervention appears to have a stimulating effect. Recurrence is local or in nearby nerves. Internal metastasis occurs late.

In all those cases in which a sarcoma was found associated with multiple neurofibromas at the time of the first examination, the authors

12. Villegas, R. R., and Rojas, P.: Consideraciones sobre los tumores primitivos de los nervios. A propósito de un fibrosarcoma de la vaina del ciático, Semana méd. **27**:675, 1920.

13. McGuire, E. R., and Burden, J. F.: Unusual Case of Sarcoma of the Median Nerve, Surg. Gynec. Obst. **35**:453, 1922.

14. Meyer, E.: Zur Kenntnis der Rueckenmarkstumoren, Deutsche Ztschr. f. Nervenh. **22**:232, 1902.

15. Kredel, L., and Beneke, R.: Ueber Ganglioneurome und andere Geschwuelste des peripheren Nervensystems, Deutsche Ztschr. f. Chir. **67**:239, 1902.

16. Buford, C. G., and Davis, T. A.: Sarcoma of the Sciatic Nerve, Surg. Gynec. Obst. **20**:495, 1915.

17. Allenbach, E.: Le sarcome du nerf sciatique, Rev. de chir. **59**:134, 1912.

18. Garrè, C.: Ueber sekundaer maligne Neurome, Beitr. z. klin. Chir. **9**: 465, 1892.

concluded from the symptomatology that one of these neurofibromas had undergone a sarcomatous change. According to Garrè, there is a striking tendency to sarcomatous transformation in multiple neurofibromatosis, for in at least 12 per cent of all cases this sarcomatous change takes place from a benign neurofibroma. Garrè calculated this percentage on the basis of seventeen collected cases. From table 1, it is seen that up to 1927 there were reported fifty-nine cases of sarcomatous transformation in von Recklinghausen's disease. Using the 1927 figures of Fischer,[19] when he collected reports of 466 cases of von Recklinghausen's disease, one obtains an incidence of malignant changes of about 13 per cent which is in agreement with Garrè. These neurosarcomas have been observed most frequently in the deeper nerve trunks. Ewing [20] stated that the serious nature of these deeper tumors is very imperfectly realized by most surgeons, so that the number of victims of this disease is much larger than is generally believed. He added that the primary attack on these tumors must be undertaken with great caution. Stromeyer [21] performed a disarticulation for a sarcoma of the median nerve which had invaded the adjacent structures. In de Morgan and Coupland's case,[22] a plexiform neurofibroma of the musculospiral nerve, associated with elephantiasis of the skin of the forearm, became sarcomatous. The musculocutaneous nerve was involved by the sarcomatous growth in Vangehr's case.[23] Pomorski [24] described a large intrathoracic tumor which compressed the ascending vena cava, causing hydrops, ascites and anasarca. The clavicular tumor in Modrzejewski's case [25] suddenly began to grow rapidly, reaching the size of a man's head, and became very painful. Westphalen [26] reported a case of multiple fibromas of the skin, nerves and ganglions, one of which in the fifth cervical nerve underwent a sarcomatous change.

---

19. Fischer, G. A.: Recklinghausensche Krankheit und Muttermaeler, Dermat. Wchnschr. **84**:89, 1927.

20. Ewing, J.: Neoplastic Diseases, ed. 2, Philadelphia, W. B. Saunders Company, 1922, p. 152.

21. Stromeyer: Handbuch der Chirurgie, 1844, vol. 1, p. 413; cited by Garrè (footnote 18).

22. De Morgan, C., and Coupland: Case of Multiple Neuroma of the Forearm, Tr. Path. Soc. London **26**:*2*, 1875.

23. Vangehr, A.: Inaug.-Diss., Munich., 1881; cited by Garrè (footnote 18).

24. Pomorski, J.: Ein Fall von Rankenneurom der Intercostalnerven mit Fibroma molluscum und Neurofibromen, Virchows Arch. f. path. Anat. **111**:60, 1888; Inaug.-Diss., Greifswald, July 30, 1887.

25. Modrzejewski, E.: Multiple angeborene Fibromata mollusca (sogenannte Neurofibrome von Recklinghausen's), Berl. klin. Wchnschr. **19**:627, 1882.

26. Westphalen, H.: Multiple Fibrome der Haut, der Nerven und Ganglien mit Uebergang in Sarcom, Virchows Arch. f. path. Anat. **114**:29, 1888.

In their cases of multiple neurofibromatosis, Campana [27] described a sarcomatosis of the skin, Sorger [28] a sarcoma of the axilla, von Gernet [29] a sarcomatous transformation of a massive plexiform fibroma of the shoulder and upper part of the arm, Heydweiller [30] a fibrosarcoma of the right popliteal space, Feindel [31] a sarcome globocellulaire of the leg, and Gluge [32] a sarcoma in the pelvis, the size of a child's head. Rolleston [33] described a sarcomatous growth involving the brachial plexus which occupied the anterior mediastinum on the left side, obliterated the internal jugular vein at its termination, invaded the structures of the neck and left lung and eroded the bodies of the fifth, sixth and seventh cervical vertebrae. The abdominal sympathetics around the aorta showed nodular fibromatous thickenings. In Cimmino's case,[34] a large sarcoma in the sacral region was associated with about 450 fibromatous tumors of the skin. Jeanselme and Orrillard [35] reported a case of fibrosarcoma of the saphenous nerve with elephantiasis molle congenita of the overlying skin. Adrian [36] found at necropsy a fibrosarcoma of the duodenum which was associated with multiple fibromas of the skin and internal viscera and a neuromyxoma in the supraclavicular fossa about the size of a child's fist. In MacKenzie's case,[37] in addition to the multiple soft tumors in the skin, a goose egg-sized sarcoma of the sciatic nerve was observed. In Kohtz's case,[38] besides the numerous nodules on the anterior and

27. Campana: Ueber Sarkomatose der Haut: XII. Kongress der ital. Aerzte zu Pavia, Sept. 19-25, 1887, Sektion fuer Syphilis u. Hautkrankheiten, Monatsh. f. prakt. Dermat. **7**:30, 1888.

28. Sorger, J. F.: Beitrag zur Lehre von den multiplen Neurofibromen, Inaug.-Diss., Erlangen, Dec. 22, 1890, p. 31.

29. von Gernet, R.: Das plexiforme Fibrom der nerven und der Haut. Ein Beitrag zur Geschwulstlehre, Inaug.-Diss., Dorpat, April 4, 1892, p. 23.

30. Heydweiller, E.: Ein Beitrag zur Casuistik der multiple Fibrombildungen der Haut (Mollusca fibrosa), Inaug.-Diss., Erlangen, Dec. 30, 1887, p. 28.

31. Feindel, E.: Sur quatre cas de neurofibromatose généralisée, Thèse de Paris, 1896, no. 104, p. 76.

32. Gluge, G.: Abhandlungen zur Physiologie und Pathologie, 1841, p. 140, cited by Garrè (footnote 18).

33. Rolleston, H. D.: A Case of Recklinghausen's Disease Complicated with a Sarcomatous Growth Involving the Brachial Plexus, Lancet **2**:271, 1899.

34. Cimmino, R.: Su di un caso di fibromi multipli cutanei con metamorfosi sarcomatosa, Gior. ital. d. mal. ven. **26**:28, 1891.

35. Jeanselme, E., and Orrillard: Contribution à l'étude des malformations congénitales de la peau et de l'hypoderme, Rev. de chir. **14**:50, 1894.

36. Adrian, C.: Ueber einen bemerkenswerthen Fall von Neurofibromatosis, Wien. klin. Wchnschr. **15**:813, 1902.

37. MacKenzie, K. A. J.: Resection of the Sciatic Nerve, Ann. Surg. **50**:295, 1909.

38. Kohtz: Ein Fall von multiplen Fibromen der Haut, Inaug.-Diss., Koenigsberg, March 10, 1893, p. 28.

cardiac surfaces of the stomach and several subserous nodules in the small intestines and the mesentery, there was a sarcomatous tumor of the duodenum. Shouldice [39] reported a case of multiple peripheral fibromas in which operation disclosed dozens of small tumors about the size of millet seeds all over the stomach and a fibromyxosarcoma on the greater curvature of the stomach. Wright [40] noted a case of familial neurofibromatosis in which a sarcomatous change took place in one of these tumors in the thigh. Feriz's case [41] showed molluscum fibrosum, elephantiasis neuromatosa of the skin of the right leg, and a large fibrosarcoma of the lateral aspect of the thigh. In a case of multiple peripheral neurofibromatosis, Hartman [42] described a large fibromyxosarcoma of the stomach which he believed arose presumably in a neurofibroma of the gastric wall. In one of Gray's cases,[43] necropsy showed a spindle cell sarcoma of the mediastinum.

It has been clinically observed that partial removal or any operative trauma of a neurofibroma may activate the tumor into a sarcoma. In Arnozan's first case,[44] a plexiform neurofibroma of the right side of the neck became sarcomatous after the operation and gradually increased in size during an interval of fifteen years, measuring 40 by 10 by 10 cm. and weighing 1,850 Gm. The pedunculous tumor masses finally became gangrenous and the patient died from septicemia. Goldmann [45] removed a fibromyxoma of the tenth intercostal nerve extending from the level of the eighth dorsal to that of the first lumbar vertebra. A year later, the patient returned with an equally large tumor, recurrent in the operative scar. After the second operation, the second recurrence grew rapidly, reaching the size of a child's head within two months. Microscopic examination of the recurrent tumors showed a spindle cell sarcoma. There was no internal metastasis

---

39. Shouldice, E.: Multiple Fibromata—A Case With One Fibroma Enlarging Into the Stomach, Canad. M. A. J. **15**:66, 1925.

40. Wright, A., quoted by Shouldice (footnote 39).

41. Feriz, H.: Neurofibromatosis und Sarkom, Deutsche Ztschr. f. Chir. **192**: 372, 1925.

42. Hartman, H.: Fibro-Myxo-Sarcoma of the Stomach in a Case of Neurofibromatosis (von Recklinghausen's Disease), Surg. Gynec. Obst. **44**:308, 1927.

43. Gray, S. H.: The Histogenesis of von Recklinghausen's Disease, Arch. Neurol. & Psychiat. **22**:91 (July) 1929.

44. Arnozan, X.: Névrome plexiforme, J. de méd. de Bordeaux **15**:72, 1885; Trois cas de névrome plexiforme dans le recueil d'observations de dermatologie, Bordeaux, 1892. Meslet: Contribution à l'étude des névromes plexiformes, Inaug. Diss., Bordeaux, 1892, no. 6.

45. Goldmann, E. E.: Beitrag zu der Lehre von den Neuromen, Beitr. z. klin. Chir. **10**:13, 1893.

at necropsy. In Delore and Bonne's case,[46] a cellular neurofibroma developed on the exact spot where a violent blow had been received three years previously. There were two operations inside of three months, the second being for recurrence. Ten months later, there was a second recurrence. Harbitz [47] reported fourteen cases of von Recklinghausen's disease, in two of which there occurred a sarcomatous transformation in a preexisting fibromatous nodule. In one of these cases, a fibroma of a thigh nerve underwent sarcomatous changes after ten repeated attempts at extirpation in the course of eighteen years. In the second case, a plexiform neurofibroma of the left sciatic nerve after repeated removals and recurrences finally became sarcomatous. In my first case, an edematous fibroma of the lower cervical and upper mediastinal regions at the second operation about one year later had already become a very cellular spindle cell sarcoma.

Furthermore, even after complete extirpation of a sarcomatous tumor, another neurofibroma in a different location may undergo sarcomatous transformation. In von Winiwarter's case,[48] it is interesting to note that the operative removal of even a benign neurofibroma in the left scapular region was soon followed by a sarcomatous transformation of the preexisting neurofibroma of the left radial nerve. Finotti [49] reported a case of fibroma durum epineuricum of the great sciatic nerve; when the patient was seen again about eleven months after the operation, there was no recurrence at the site of the operation, but on the posterior aspect of the leg close to the lower part of the popliteal space, a fibrosarcomatous nodule was removed. Hartmann [50] excised a fibrosarcoma of the right axilla. In the course of a year, a tumor the size of a goose egg appeared in the right groin, which after operative removal was found to be a fibroma in the lower part of the tumor, calcified in the central portions, and a spindle cell sarcoma in the upper part. In Habermann's case,[51] about a year after the removal of a fibrosarcoma of the right sciatic nerve, another fibrosarcoma developed in the right ulnar nerve which was removed and the nerve sutured

46. Delore, X., and Bonne, C.: Neuro-fibromatose et névrome plexiforme, Gaz. hebd. des méd. et de chir. **45**:145, 1898. Delore: Neuro-fibromatose cutanée avec xanthome profond du bras droit, Gas. d. hôp. **69**:514, 1896.

47. Harbitz, F.: Multiple Neurofibromatosis (von Recklinghausen's Disease), Arch. Int. Med. **3**:32 (Feb.) 1909.

48. von Winiwarter, A.: Plexiformes Fibro-neurom der Armnerven mit circumscripter Hauthypertrophie und Sarcomentwickelung, Arch. f. klin. Chir. **19**:595, 1876.

49. Finotti, E.: Beiträge zur Chirurgie und pathologischen Anatomie der peripherischen Nerven, Virchows Arch. f. path. Anat. **143**:133, 1896.

50. Hartmann, H.: Zur Kenntnis der sekundaer malignen Neurome, Beitr. z. klin. Chir. **17**:177, 1896.

51. Habermann: Beitrag zur Kenntnis der secundaer malignen Neurome, München. med. Wchnschr. **45**:713-716; 752-754, 1898.

together. About nine months later, painful nodules developed in the left occipital region, which on removal showed beginning fibrosarcoma. There were no local recurrences after each of the three operations. In Thomson's case, a rapidly growing tumor of the right median nerve was removed, which proved to be a spindle cell sarcoma at one point in the sections. The sarcoma recurred twice, each followed by an operation, the last being a shoulder amputation. It recurred for the third time in the vicinity of the scar. Necropsy revealed the unexpected finding of a spindle cell sarcoma of the left lumbar plexus, about the size of a child's head, involving the psoas muscle and the adjacent bodies of the vertebrae. In Herxheimer and Roth's first case, there were multiple neurofibromas of the peripheral nerves, one of which in the anterior thigh became sarcomatous. Soon after its operative removal, a nodule in the neck began to enlarge rapidly, causing marked compression of the trachea. Necropsy showed that the sarcomatous tumor in the neck had extended into the left thoracic cavity, where it was adherent to the apex of the lung. There was also a metastatic nodule in the right lung.

Recurrences may be frequent and are generally local or regional after operative measures. Such recurrences may be repeated several times before distant metastasis takes place. In fact, the absence of metastasis is remarkable in the reported cases in which there were repeated operations for recurrences. Hitchcock [52] removed a sarcoma of the ulnar nerve which recurred. Amputation at the shoulder joint was performed, which was followed by a second recurrence in the shoulder. There was no necropsy. In Girard's first case,[53] three operations were performed for the original tumor and the two recurrences. The patient was still living at the last report. Volkmann,[54] Virchow [55] and Blasius [56] separately reported the progress notes of several interesting cases. In the case of Volkmann and Blasius, the patient had three operations for a tumor of the median nerve during the course of eight years; each time there was a recurrence. At necropsy, there was no metastasis. In the case of Virchow and Blasius the patient had six operations for the original tumor, with five recurrences. There was no

52. Hitchcock, A.: Some Remarks on Neuroma with a Brief Account of Three Cases of Anomalous Cutaneous Tumors in One Family, Am. J. M. Sc. **43**:320, 1862.

53. Girard: Transformation de molluscums et de naevi en sarcome et en épithéliome, Dauphine méd. **22**:1, 1898.

54. Volkmann, R.: Ueber ein faustgrosses ulceriertes Neurom im Handteller, Virchows Arch. f. path. Anat. **12**:27, 1857.

55. Virchow, R.: Ueber einen Fall von vielfachen Neuromen (sogenannten Faser-Kern-geschwuelsten) mit ausgezeichneter localer Recidivfaehigkeit), Virchows Arch. f. path. Anat. **12**:114, 1857.

56. Blasius, E.: Ueber rueckfaellige Neurome, Arch. f. klin. Chir. **2**:188, 1862; Ueber rueckfaellige Neurome, ibid. **6**:775, 1865.

necropsy. In the case of Blasius, the patient had three operations with two recurrences. The patient was said to be entirely well about a year later. Von Bergmann [57] presented a patient before the Berlin Surgical Society, on whom operation for a spindle cell sarcoma of the left axillary and scapular regions was followed by a recurrence and rapid growth. As mentioned previously, in the cases of Arnozan and of Goldmann, a plexiform neurofibroma and a fibromyxoma respectively recurred as sarcomas after operative intervention. At necropsy, there was no internal metastasis in either of these cases. Thilow [58] reported two cases, in one of which there were recurrences nearby. In Tichoff and Timofejeff's case,[59] three operations were performed for a spindle cell sarcoma of the popliteal space with two recurrences. No metastasis was found at necropsy. Scheven [60] described a spindle cell sarcoma of the median nerve; four operations were performed because of recurrences. Necropsy did not reveal any internal metastasis. Vallas [61] presented a patient with generalized neurofibromatosis who showed a recurrence in the scar, and who had already been operated on five or six times for a large tumor of the eighth intercostal nerve.

From table 1 it is seen that metastasis is relatively infrequent when a benign neurofibroma becomes sarcomatous. There were fourteen metastatic cases among a total of sixty-five, or 22 per cent. Metastasis occurred eleven times in the lung, four times in the pleura, twice in the diaphragm and the liver and once each in the humerus, pubic bone and paravertebral lymph node. It is to be noted, however, that once malignancy has supervened, the prognosis is generally bad. The patients succumb to the progressive cachexia resulting from the many recurrences, metastasis and repeated operations. Busch [62] performed an amputation of the leg for a sarcoma of the peroneal nerve with recurrence and invasion of the bone. Necropsy showed numerous sarco-

57. von Bergmann: Presentation of Case, Verhandl. d. Freie Vereinigung der Chirurgien Berlins, Sitzung Nov. 18, 1889, koeniglichen Klinikum, Berl. klin. Wchnschr. **26**:1133, 1889.

58. Thilow, C.: Beiträge zur Casuistik des Fibroma molluscum multiplex, Inaug.-Diss., Berlin, Oct. 25, 1889. The same case was presented by Schlange before the Freie Vereinigung der Chirurgien Berlins, Jan. 14, 1889, koeniglichen Klinikum, Berl. klin. Wchnschr. **26**:122, 1889.

59. Tichoff and Timofejeff: Multiple Neurofibroma with Fibroma Molluscum Multiplex, Chir. Ann. (Russian), 1894, p. 719; abstr., Jahr. ü. d. Leistung. in d. ges. Med., XXIX. Jahrgang, Bericht für das Jahr 1894, Berlin, A. Hirschwald, 1895, vol. 2.

60. Scheven, O.: Zur Kenntnis der sekundaer malignen Neurome, Beitr. z. klin. Chir. **17**:157, 1896.

61. Vallas: Neuro-fibromatose généralisée, Soc. de Chir. de Lyon, June 29, 1899, Lyon méd. **92**:132, 1899.

62. Busch: Handbuch der allgemeinen und speciellen Chirurgie, Erlangen, Pitha & Billroth, 1865, vol. 2, p. 239; cited by Garrè (footnote 18).

matous nodules along the intercostal nerves and lungs. In Genersich's case,[63] necropsy showed that the right sciatic sarcoma had produced metastatic nodules in both lungs, in several of which the arteries were occluded with fibrin and spindle cells. In two of the cases reported by Rose,[64] a myxosarcoma of the sciatic nerve and a retroperitoneal sarcoma of the crural nerve metastasized to the lung and to a paravertebral lymph node, respectively. Hume observed in a case of molluscum fibrosum a sarcoma of the great sciatic nerve with metastasis to the pleura, left lung, liver and left humerus. Westphalen [65] reported an unusual case of spindle cell sarcoma of the sciatic nerve located in the popliteal space, which in spite of the disarticulation of the hip joint at the very first operation had already metastasized to the lung and pleura. Poncet [66] performed an amputation for sarcoma of the thigh, but the patient succumbed to a generalized sarcomatosis. Adrian, in 1901, reported an interesting case of fibromyxosarcoma growing in the region of a meningocele, with metastasis to the diaphragm and lung. In a case of multiple neurofibromatosis, Potter and McWhorter [67] recently described a spindle cell sarcoma over the occiput which perforated the skull and formed a thick plaque over the dura, and which also metastasized to the visceral pleura and lung.

Primary multiple sarcomas arising in two or more neurofibromas in von Recklinghausen's disease appear to be a rare phenomenon. In von Recklinghausen's own case, two of the largest nodules on the jejunum were sarcomatous. Seitz' case [68] showed multiple tumors of the skin and nerves, the largest of which was in the left peroneal nerve and was about the size of a fist. There was a total of twenty-two nodular tumors of the various nerves of the body. Seitz believed his case to be one of multiple round and spindle cell sarcomas. In Arnozan's second case, a congenital plexiform neurofibroma of the right side of the neck became a sarcome fasciculé within six months following a blow, and recurred after removal. Another small pedunculated fibroma on the patient's back on removal showed early sarcomatous changes. This

63. Genersich, A.: Multiple Neurome, Virchows Arch. f. path. Anat. **49**:15, 1870.

64. Rose, E.: Ueber die Exstirpation gutartiger Bauchgewaechse (Die Laparektomie), Ztschr. f. Chir. **19**:24, 1884; Ein Neurom der Erb'schen Plexuswurzeln, ibid. **24**:392, 1886.

65. Westphalen, H.: Multiple Fibrome der Haut und der Nerven mit Uebergang in Sarcom und mit Metastasenbildung, Virchows Arch. f. path. Anat. **110**:29, 1887.

66. Poncet, A.: Discussion in Tr. Société nationale de médicine, March 29, 1897, Lyon méd. **84**:520, 1897.

67. Potter, P. C., and McWhorter, J. E.: Von Recklinghausen's Disease with Sarcomatous Degeneration of a Deep Fibroma, Ann. Surg. **90**: 397, 1929.

68. Seitz, J.: Multiple Fibrosarkome der Nerven und Perichondritis laryngea, Virchows Arch. f. path. Anat. **52**:114, 1871.

TABLE 1.—*Sarcomatous Transformation in Neurofibromatosis*

No.	Author	Sex	Age	Location of Sarcoma	Operation	Local Recurrence	Internal Metastasis	Result
1.	Gluge, 1841	M	34	Pelvis	.....	.....	.....	?
2.	Stromeyer, 1844	M	19	Median nerve	Yes	.....	.....	.....
3.	Volkmann, 1857 Blasius, 1862	F	19	Median nerve	Yes, 3	Yes, 3	None	Died
4.	Virchow, 1857 Blasius, 1862	M	56	Wrist	Yes, 6	Yes, 5	.....	Died
5.	Blasius, 1862	M	53	Arm	Yes, 3	Yes, 2	.....	Living
6.	Hitchcock, 1862	M	40	Ulnar nerve	Yes, 2	Yes, 2	? (no necropsy)	Died
7.	Busch, 1865	..	..	Peroneal nerve	Yes, 2	Yes, 2	Lung, pleura	Died
8.	Genersich, 1870	M	22	Sciatic nerve	.....	.....	Lung	Died
9.	Seitz, 1871	M	48	Peroneal nerve	No	.....	No	Died
10.	De Morgan and Coupland, 1875	F	15	Musculospiral nerve	Yes	?	.....	Living
11.	Von Winiwarter, 1876	M	36	Radial nerve	Yes	.....	Lung	Died
12.	Vangehr, 1881	F	42	Musculocutaneous nerve	Yes	.....	.....	.....
13.	Von Recklinghausen, 1882	F	55	Jejunum	No	No	No	Died
14.	Modrzejewski, 1882	F	37	Clavicular region	.....	.....	No	Died
15.	Rose, 1884	M	27	Crural nerve	Yes	No	Paravertebral lymph node	Died
16.	Arnozan, 1885	F	31	Right side of neck	Yes	Yes	No	Died
17.	Hume, 1886	M	37	Sciatic nerve	Yes	.....	Pleura, lung, liver humerus	Died
18.	Rose, 1886	M	27	Sciatic nerve	Yes	Yes	Lung	Died
19.	Pomorski, 1887	M	28	Mediastinum	No	.....	No	Died
20.	Heydweiller, 1887	M	45	Popliteal space	Yes	No	No	Died
21.	Campana, 1887	..	..	Extremity	.....	.....	.....	.....
22.	Westphalen, 1887	F	35	Sciatic nerve	Yes	.....	Lung, pleura	Died
23.	Westphalen, 1888	F	45	Fifth cervical nerve	No	.....	No	Died
24.	Von Bergmann, 1899	M	54	Axillary and scapular regions	Yes	Yes	.....	?
25.	Thilow, 1889	M	15	Intercostal nerve ?	No	.....	No	Died
26.	Thilow, 1889	M	55	Axilla	Yes	Yes	.....	Living
27.	Sorger, 1890	M	27	Axilla	Yes	.....	.....	Living
28.	Cimmino, 1891	M	42	Sacral region	Yes	.....	.....	?
29.	Garrè, 1892	F	31	Sciatic nerve	Yes	Yes	Suspicious, no necropsy	Died
30.	Arnozan, 1892	F	32	Cervical region	.....	.....	.....	Living
31.	Von Gernet, 1892	F	43	Shoulder and region of upper arm	Yes	.....	.....	Living
32.	Kohtz, 1893	F	Adult	Wall of duodenum	.....	.....	.....	Necropsy specimen
33.	Goldmann, 1893	F	54	Tenth intercostal nerve	Yes, 2	Yes, 2	No	Died
34.	Jeanselme and Orrillard, 1894	M	21	Saphenous nerve	Yes	.....	.....	Living
35.	Tichoff and Timofejeff, 1894	M	42	Popliteal space	Yes, 3	Yes, 2	No	Died
36.	Feindel, 1896	M	58	Leg	Yes	.....	.....	Living
37.	Finotti, 1896	M	44	Sciatic nerve	Yes	Yes	Yes	Died
38.	Scheven, 1896	M	24	Median nerve	Yes, 4	Yes, 3	No	Died
39.	Hartmann, 1896	F	48	Crural nerve	Yes	.....	.....	?
40.	Poncet, 1897	M	50	Thigh	Yes	.....	Yes	Died
41.	Habermann, 1898	F	28	Sciatic nerve	Yes	.....	.....	Living
42.	Delore and Bonne, 1898	M	34	Radial nerve	Yes, 2	Yes, 2	.....	?
43.	Girard, 1898	F	50	Thigh	Yes, 3	Yes, 2	.....	Living
44.	Girard, 1898	F	40	Hypochondrial region	Yes	.....	.....	Living
45.	Vallas, 1899	M	70	Intercostal nerve	Yes, 6	Yes	.....	Living?
46.	Rolleston, 1899	M	32	Brachial plexus	Yes	.....	No	Died
47.	Thomson, 1900	M	28	Median nerve	Yes, 2	Yes, 2	No	Died
48.	Adrian, 1901	F	36	Occiput	No	.....	Lung, diaphragm	Died
49.	Adrian, 1902	F	56	Duodenum	Yes	.....	No	Died
50.	Hulst, 1904	F	19	Cerebellopontine angle	.....	.....	No	Died
51.	Hulst, 1904	F	30	Intercostal nerve	.....	.....	No	Died
52.	Harbitz, 1909	F	44	Nerve of thigh	Yes, 10	Yes, many	.....	Died
53.	Harbitz, 1909	F	32	Sciatic nerve	Yes, many	Yes, many	.....	Died
54.	MacKenzie, 1909	M	45	Sciatic nerve	Yes	.....	.....	Living
55.	Herxheimer and Roth, 1914	M	38	Nerve of thigh	Yes	.....	Lung	Died

TABLE 1.—*Sarcomatous Transformation in Neurofibromatosis—Continued*

No.	Author	Sex	Age	Location of Sarcoma	Operation	Local Recurrence	Internal Metastasis	Result
56.	Herxheimer and Roth, 1914	M	55	Lumbar plexus	.....	.....	No	Died
57.	Wright, 1916.....	M	Adult	Nerve of thigh	.....	.....	.....	Died
58.	Shouldice, 1925.....	F	41	Stomach	Yes	.....	.....	Living
59.	Feriz, 1925.....	M	64	Thigh	Yes	.....	.....	.....
60.	Hartman, 1927.....	M	48	Stomach	Yes	.....	Liver, diaphragm	Died
61.	Plenge, 1928.....	M	15	Neck	Yes	.....	Lung	Died
62.	Gray, 1929.....	M	37	Mediastinum	.....	.....	No	Died
63.	Gray, 1929.....	M	25	Sciatic nerve	.....	.....	Lung, pubic bone	Died
64.	Potter and McWhorter, 1929	M	22	Occiput	Yes	Yes	Lung, pleura	Died
65.	Hosol, 1930.....	M	28	Lower cervical and upper mediastinal regions	Yes, 2	No	Suspicious, no necropsy	Died

case illustrates very well the low grade and local nature of the malignant growth, since the patient has been carrying the recurrent growth for the past fifteen years. In Hulst's first case,[69] there was a bilateral fibrosarcoma of the cerebellopontile angle as well as multiple intracranial neurofibromas. In a case of plexiform neurofibromas of the neck, Plenge[70] described a spindle cell sarcoma located in the region of the atlas and a mixed cell sarcoma in the region of the submaxillary gland. Does this rarity mean that the presence of a sarcoma has an inhibiting influence on the development of another sarcoma elsewhere in the nervous system? As stated previously, the removal of one sarcomatous growth may be followed by a sarcomatous transformation of another neurofibroma distantly located. Indeed, Cramer[71] has noted in his studies on experimental carcinogenesis that it was rare to find a simultaneous development of malignancy in two or three distant centers of the same area, while it was not uncommon to obtain a successive development in two or even three distant centers after the operative removal of the preexisting malignant tumor. According to his conception of potential malignancy, a large area of cells in a given tissue may be in a condition in which they do not give any manifestation of malignancy either as judged from their histologic appearance or from their biologic behavior, but may, nevertheless, be particularly prone to the development of malignancy when the local inhibitory factors are broken down or shifted. It is interesting to note that Cramer has been able to break or shift these local inhibitory factors by applying a cautery or a caustic pencil to the base of one of the benign growths, which then became malignant in six of the fourteen experiments. Clinically, this

69. Hulst, J. P. L.: Beitrag zur Kenntnis der Fibrosarkomatose des Nervensystems, Virchows Arch. f. path. Anat. **177**:317, 1904.

70. Plenge, K.: Ueber eine eigenartige plexiforme Neubildung des Nervensystems in der Gegend des Halses und der Halswirbelsaeule, Virchows Arch. f. path. Anat. **269**:83, 1928.

71. Cramer, W.: On Experimental Carcinogenesis: The Local Resistance of the Skin to the Development of Malignancy, Brit. J. Exper. Med. **10**:335, 1929.

same thing is seen in certain cases when a benign neurofibroma is traumatized by operative procedures.

Cases are on record in which operative removal of a painful neurofibromatous nodule may be followed by crops of multiple neurofibromas elsewhere in the body. In spite of repeated operations, no sarcomatous changes occur, as shown by microscopic examination. It is still unaccountable why in some cases a malignant transformation supervenes after a single operation and in other cases even after repeated operations no malignant changes arise. At the age of 36, von Buenger's [72] patient noticed for the first time a painful tumor the size of a pigeon egg on the right thigh. Operation showed this to be a neurofibroma of the nervus cutaneus femoris externus. Gradually, small numbers of painful nodules began to develop along the course of the nerves of the right thigh. A symptomless neurofibroma also developed in the neck, and was removed. At another operation, all the involved nerves in the right thigh were removed in toto. Several months later, similar multiple nodules developed in the other thigh. On removal these were found to be pure neurofibromas. Soon multiple tumors began to appear over the entire body. The patient developed signs of increasing intraspinal pressure and a terminal bronchopneumonia. Necropsy showed that all the spinal nerve roots without exception, the peripheral nerves and the larger part of the sympathetics were involved in the same neurofibromatous process. Microscopically, no malignant changes were found anywhere. This fibromatosis of the sympathetic nerves and ganglions has frequently been observed at necropsy in cases of von Recklinghausen's disease. It has been found that the nature of this enlargement in the sympathetics is precisely similar to that occurring in the cerebrospinal nerves.

From the literature, a total of 65 cases of sarcomatous transformation in von Recklinghausen's disease has been collected. There were 39 male to 24 female patients, or a ratio of 1.6:1. The sex was not stated in 2 cases. As has been pointed out by Alvarez and myself,[73] every sex ratio should be corrected by a factor dependent on the sex distribution of the group of men and women studied. Fortunately, Fischer, in 1927, collected 466 cases of von Recklinghausen's disease, of which 299 were men and 167, women, which gives a sex distribution of 1.8:1. Now, since the sex distribution of all cases of von Recklinghausen's disease and of those cases of this disease showing malignant changes is practically the same, it can be statistically stated that sex is of no etiologic importance in cases of sarcomatous transformation. Table 2

72. von Buenger, O.: Ueber allgemeine multiple Neurofibrome des peripherischen Nervensystems und Sympathicus, Arch. f. klin. Chir. **55**:558, 1897.

73. Hosoi, K., and Alvarez, W. C.: The Influence of Sex on the Incidence of Gastro-Intestinal Disease, Human Biol. **1**:63, 1930.

shows the age incidence. Seventy-two per cent of the cases occurred in the third, fourth and fifth decades. The youngest was 15 years of age, the oldest 70 years. The age of onset of von Recklinghausen's disease ranged from birth to about 25 years.

In résumé, malignant transformation takes place in about 13 per cent of all cases of von Recklinghausen's disease. When this happens, the tumor grows rapidly and tends to recur locally even after repeated operative measures. Metastasis is usually late and frequently absent, but may rarely occur early. In some cases, the mere extirpation of a malignant tumor or even a benign one appears to stimulate another neurofibroma distantly located to undergo sarcomatous transformation. Prognosis is very poor, the patient succumbing to cachexia and post-operative complications if not to the metastasis.

It is well to bear in mind that not all peripheral multiple tumors in the skin are mesodermal in origin. A few cases have been reported in which multiple ectodermal tumors in the skin closely simulated

TABLE 2.—*Percentage of Age Distribution in Malignant Transformation*

Age periods in years	11-20	21-30	31-40	41-50	51-60	61-70
Percentage	10	21	26	25	15	3

von Recklinghausen's disease. In Anderson and Shennan's case,[74] the entire face and both shoulders of a 12 weeks old infant were closely studded over with minute growths which proved to be sebaceous cysts. A more striking case is that of Ferreira and Garaude,[75] who actually counted 276 sebaceous cysts scattered all over the body of a 65 year old man including the head and both extremities. That adenoma sebaceum may occur intermingled with mollusca fibrosa is shown in the case of Anderson[76] and others. Another type of tumor, neuro-epithelial in origin and hence ectodermal, may produce a more or less striking resemblance to von Recklinghausen's disease. These are the true neuromas of Virchow in contrast to the false neuromas or neuro-fibromas. In a case of neuroma verum multiplex amyelinicum gangliosum, Knauss[77] counted more than 60 of these multiple cutaneous nodules; many more were said to be present. Kredel and Beneke[15] counted about 160 of these cutaneous nodules in their case of multiple

74. Anderson, J. S., and Shennan, T.: A Neuroblastoma of the Thoracic Cavity, J. Path. & Bact. **26**:545, 1923.

75. Ferreira, O., Jr., and Garaude, P.: Kystos sebaceous generalizados simulando a doenca de Recklinghausen, Brasil-méd. **2**:343, 1926.

76. Anderson, W.: A Case of Adenoma Sebaceum Intermingled with Mollusca Fibrosa, Brit. J. Dermat. **7**:316, 1895.

77. Knauss, K.: Zur Kenntnis der aechten Neurome. Neuroma verum multiplex amyelinicum gangliosum, Virchows Arch. f. path. Anat. **153**:29, 1898.

ganglioneuroma of the sympathetics. Among the dermatologists, Levin and Tolmach [78] and recently Ludy [79] pointed out that the atypical form of cutaneous neurofibromatosis (those occurring on the extremities and adjacent regions) may be so closely simulated by sarcomas, sarcoids of Boeck-Darier-Roussy, tuberculids and other skin diseases that it is frequently incorrectly diagnosed clinically.

Because of its multiplicity in numbers and its generalized distribution, because of its very frequent association with a multiplicity of congenital abnormalities, and because of its frequent familial character, von Recklinghausen's disease is now generally looked on as a congenital anomaly of the nervous system in a hereditary and dysontogenetic sense. In his review of 466 cases of von Recklinghausen's disease described in the literature, Fischer could find no definite relationship between it and the nevi or pigmented patches. For this reason, the use of the term "incomplete neurofibromatosis" by some dermatologists when only the café au lait spots or melanoderma are present in familial cases is not justified. Levin [80] and Tucker [81] believed that a definite etiologic relationship existed between endocrine dysfunction and von Recklinghausen's disease. The cases of Kawashima [82] and of Herxheimer and Roth were associated with a paraganglioma of the suprarenal gland. On the contrary, Schneiderman [83] stated that a study of his cases failed to reveal any endocrine dysfunction in the etiology of von Recklinghausen's disease, and that the endocrine glands should not be blamed for the occasional occurrence of tumors in them in association with the neurofibromatosis. At this point, it may be of interest to note that Brickner [84] described a fibroma molluscum gravidarum in which as many as 40 or 50 sessile and pedunculated fibromas may appear during pregnancy and disappear as rapidly soon after delivery. Lehman [85] made a plea for a more complete roentgenographic study of

78. Levin, O. L., and Tolmach, J. A.: The Atypical Form of Neurofibroma: Its Differential Diagnosis from Other New Growths of the Skin, New York State J. Med. **27**:819, 1927.

79. Ludy, J. B.: Cutaneous Neuroma, Arch. Dermat. & Syph. **21**:419 (March) 1930.

80. Levin, O. L.: Recklinghausen's Disease: Its Relation to the Endocrine System, Arch. Dermat. & Syph. **4**:303 (Sept.) 1921.

81. Tucker, B. R.: Von Recklinghausen's Disease with Special Consideration of the Endocrine Connection, Arch. Neurol. & Psychiat. **11**:308 (March) 1924.

82. Kawashima, K.: Ueber einen Fall von multiplen Hautfibromen mit Nebennierengeschwulst, Virchows Arch. f. path. Anat. **203**:66, 1911.

83. Schneiderman, H.: Recklinghausen's Disease, Arch. Dermat. & Syph. **12**: 483 (Oct.) 1925.

84. Brickner, S. M.: Fibroma Molluscum Gravidarum: A New Clinical Entity, Am. J. Obst. **53**:191, 1906.

85. Lehman, E. P.: Recklinghausen's Neurofibromatosis and the Skeleton: A Plea for Complete Study of the Disease, Arch. Dermat. & Syph. **14**:178 (Aug.) 1926.

the skeleton in all cases of von Recklinghausen's disease. Are the frequently associated bone changes to be regarded as an integral part of multiple neurofibromatosis, as a consequent event or as additional evidence that some congenital influence is at work? More exact observations must be made before these questions can be definitely answered. In the first case here reported, the many roentgen examinations showed no skeletal defects. Pack [86] reported a remarkable case of neurofibromatosis of the cranial and deep cervical nerves, presumably unilateral from the symptomatology.

In addition, von Recklinghausen's disease is often associated with one or more other types of tumors. As experience has shown, such multiplicity of tumors connotes an underlying congenital defect. Winestine [87] pointed out the close relationship that may exist between von Recklinghausen's disease and giant growth and blastomatosis of the intestines. Harbitz reported an unusual case which showed at necropsy a carcinoma of the left ovary with peritoneal and pleural metastases, a large spindle cell sarcoma of the mesentery and molluscum fibrosum. In Takács' patient,[88] the neurofibromatosis was associated with a large cavernous hemangioma of the right forearm. Girard's third case is unusual in that, following trauma, the skin over a molluscum was transformed into an ulcerating epithelioma. Similar carcinomatous changes over one of the neurofibromas have been described by Schmit [89] and Chambard.[90] Patoir and Raviart [91] reported a case of three gliomas of the spinal cord with multiple neurofibromas of the nerve roots. Kaulbach's case [92] is of much interest in that the multiple neurofibromas of the peripheral nerves were associated with multiple central neurofibromas, multiple gliomatous changes in the spinal cord and a psammosarcoma of the dura mater situated on the rim of the foramen magnum.

---

86. Pack, G. T.: Unilateral Neurofibromatosis of the Cranial and Deep Cervical Nerves: Report of a Case, Arch. Neurol. & Psychiat. **21**:919 (April) 1929.

87. Winestine, F.: The Relation of von Recklinghausen's Disease (Multiple Neurofibromatosis) to Giant Growth and Blastomatosis, J. Cancer Research **8**:409, 1924.

88. Takács, A.: Ueber multiple Neurome, Virchows Arch. f. path. Anat. **75**: 431, 1879.

89. Schmidt, C.: Transformation carcinomateuse d'un molluscum, Franç. méd. **26**:481, 1879.

90. Chambard, E.: Contribution à l'étude de la transformation cancéreuse des neoplasmes bénins de la peau, Ann. de dermat. et syph. **4**:61, 1883.

91. Patoir, J., and Raviart, G.: Gliomes et formation cavitaire de la moelle. Neurofibromes radiculaires. Névrite des sciatiques. Contribution à l'étude de la lésion dite syringomyélie, Arch. de méd. exper. **13**:93, 1901.

92. Kaulbach, F. W. J.: Ein Fall von multipler Neurofibromatose des peripheren Nervensystems kombiniert mit Fibromen der Nervenwurzeln, Gliomen des Rueckenmarks und Sarkomen der Dura mater, Inaug.-Diss., Marburg, 1906, p. 39.

In a somewhat similar case, recently reported by Penfield and Young,[93] the multiple central and peripheral neurofibromas were associated with multiple tumors of the glioma series in the brain and spinal cord and with multiple meningiomas of both the dura and arachnoid. In Orzechowski and Nowicki's case,[94] in addition to the central and peripheral neurofibromatosis, there were multiple meningiomas of the dura, ependymomas of the spinal cord and medulla and tuberous sclerosis of the cerebral hemispheres. In Funkenstein's case,[95] a psammoma and an osteopsammoma of the spinal dura were associated with bilateral fibromas of the cerebellopontile angle and multiple neurofibromatosis. Régnier and his associates[96] believed that they have been unable to find microscopically all "transitions" between the multiple meningiomas and multiple tumors of the acoustic and peripheral nerves observed in their case. In a previous paper,[97] I found among twenty-two collected cases of multiple intracranial meningiomas, eight cases associated with bilateral fibromas of the acoustic nerve, four of which also showed multiple neurofibromatosis of the cranial and peripheral nerves. At that time, it was stated that the association of meningeal tumors and multiple neurofibromatosis is probably not an *a priori* relationship, but an expression of the predisposition or susceptibility of the person through hereditary and dysontogenetic influences.

SUMMARY

Two cases of von Recklinghausen's disease are here reported, in one of which an edematous fibroma of the inferior cervical and upper mediastinal regions became sarcomatous about one year after operative intervention. The microscopic picture indicates that the multiple neurofibromas originated from the perineurium. The literature on the subject of malignant transformation in multiple neurofibromatosis is reviewed.

Drs. George E. Beilby and Thomas Ordway permitted me to use the clinical histories of the cases here reported, and Dr. William P. Howard allowed me to use the x-ray plates.

93. Penfield, W., and Young, A. W.: The Nature of von Recklinghausen's Disease and the Tumors Associated with It, Arch. Neurol. & Psychiat. **23**:320 (Feb.) 1930.

94. Orzechowski, K., and Nowicki, W.: Zur Pathogenese und pathologischen Anatomie der multiplen Neurofibromatose und der Sclerosis tuberosa (Neurofibromatosis universalis), Ztschr. f. d. ges. Neurol. u. Psychiat. **11**:236, 1912.

95. Funkenstein, O.: Ein Beitrag zur Kenntnis der Tumoren des Kleinhirnbrueckenwinkels ("zentrale Neurofibromatose," "Akustikusneurome"), Mitt. a. d. Grenzgeb. Med. u. Chir. **14**:157, 1905.

96. Régnier; Dechaume, J.; Bourrat and Lesbros: Tumeurs méningées et tumeurs des nerfs craniens, Ann. d'anat. path. **4**:940, 1927.

97. Hosoi, K.: Meningiomas: With Special Reference to the Multiple Intracranial Type, Am. J. Path. **6**:245 (May) 1930.

CONGENITAL DEFICIENCY OF THE PERICARDIUM

THE FUNCTION OF THE PERICARDIUM *

CLAUDE S. BECK, M.D.
CLEVELAND

Congenital deficiency of the parietal pericardium is a rare anomaly. Sixty-four cases were collected from the literature by Moore [1] in 1925, and another case was added by Grant [2] in 1926. The case reported here is the sixty-seventh. Aside from its rarity, this condition possesses a special interest in that these cases throw some light on the function of the pericardium, a subject about which there exists considerable diversity of opinion.

REPORT OF CASE

A colored man, aged 56, was admitted to the Lakeside Hospital on Feb. 14, 1929, with the complaint of shortness of breath and swelling of the ankles. Prior to the present illness the patient's general health had been good. There was no history of shortness of breath, precordial pain or edema. The patient always had done heavy manual labor. About one month before his admission to the hospital, shortness of breath had developed. Orthopnea, a productive cough and edema of the ankles developed subsequently. Otherwise the history contained little worthy of note.

The patient was well developed and well nourished. He had serious decompensation. The systolic blood pressure was 260, and the diastolic 140 mm. of mercury. Generalized arteriosclerosis was present. The temperature was 37.5 C. (99.5 F.). The heart was somewhat enlarged, and there was a loud systolic murmur at the apex. The pulse rate was regular, 96 per minute, and after the administration of digitalis it dropped to 60 per minute. Pneumonia developed on the right side, and death occurred twelve days after admission to the hospital.

When the sternum was removed at necropsy, the heart and the left lung lay in a common serous cavity in which there was also about 1,200 cc. of clear, light yellow fluid. There was no mediastinal reflection of the parietal pleura on either side of the heart, so that the entire left ventricle was in contact with the left lung. The pericardium was almost completely absent. The ventricles were entirely free from adhesions. A fold of pericardium extended across the base of the heart from the right auricle, to which it was adherent, to the root of the left lung, where it merged with the parietal pleura. This fold was about 4 cm. broad and possessed a mesothelial surface adjacent to the heart. It was attached also by a few strands of tissue to the pulmonary artery and to the aorta (fig. 1).

---

* Submitted for publication, May 2, 1930.

* From the Department of Surgery, the Lakeside Hospital and the Western Reserve University School of Medicine.

1. Moore, R. L.: Congenital Deficiency of the Pericardium, Arch. Surg. **11**:765 (Nov.) 1925.

2. Grant, R. T.: Congenital Pericardial Deficiency: An Observation on the Function of the Pericardium, Heart **13**:371, 1926.

Extending from the hilus of the right lung to the diaphragm was a blind pouch of fibrous tissue having a mesothelial lining and almost completely covering the right auricle. In the illustration this pouch is shown opened, with its wall retracted toward the right lung. It closed off the space between the sternum anteriorly and the vertebrae posteriorly. The right phrenic nerve lay in the base of this pouch on the pulmonary aspect.

Posteriorly, there was a soft collar or pad of areolar tissue and fat extending from the hilus of the left lung across the base of the heart to the lateral aspect of the right auricle to which it was adherent. This structure ended near the junction of the diaphragm and the blind pouch referred to. The superior margin of this collar was adherent to the heart at the atrioventricular junction, and as it curved forward to its attachment to the right auricle it exerted a slight but definite

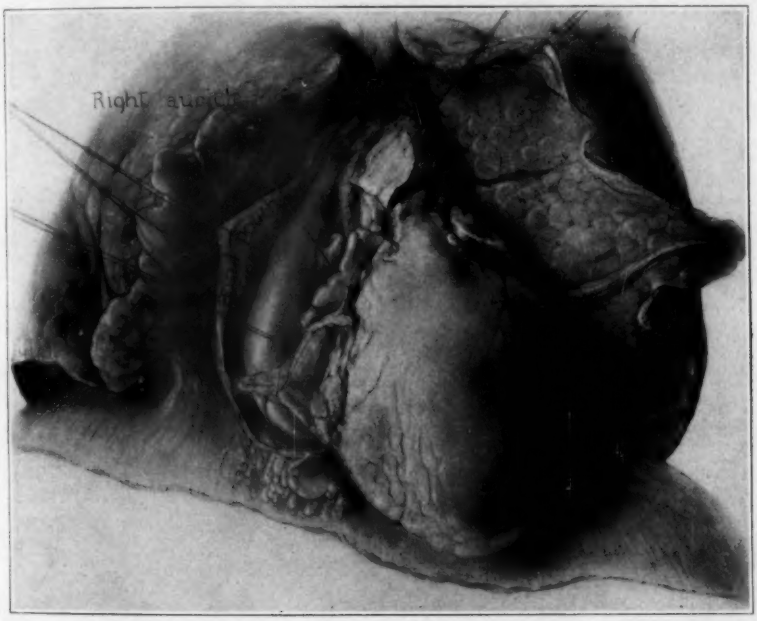


Fig 1.—Anterior view of the heart and lungs. The fold of pericardium extending over the anterior surface of the heart from the right auricle to the root of the left lung is shown cut through and the ends retracted. The blind pouch enclosing the right auricle and venae cavae is incised longitudinally and the fold of pericardium retracted toward the right lung. The right phrenic nerve lies on the pulmonary aspect of this retracted fold.

constricting force just below the tricuspid orifice. The surface of this collar of tissue was smooth and glistening (fig. 2).

The surface of the ventricles contained an increased amount of epicardial fat, and over the epicardium at the base there was an increased amount of loose areolar tissue. Toward the apex of the heart the epicardium appeared normal. The heart was free from adhesions, except along the atrioventricular groove posteriorly and the right auricle anteriorly and to the right. The heart was definitely enlarged, the enlargement consisting chiefly in hypertrophy of the left ventricle and slight

dilatation of the right auricle. After being fixed in a diluted solution of formaldehyde U. S. P. (1:10), the heart measured 10.5 cm. in its transverse diameter, 14 cm. from base to apex and 8.5 cm. from its anterior to its posterior surface. It weighed 550 Gm. The thickness of the left ventricular wall varied from 10 to 22 mm. and that of the right ventricular wall from 4 to 8 mm. The myocardium grossly showed no abnormality; microscopically there was some hypertrophy of the muscle fibers, some slight patchy fibrosis of the myocardium and some loss of nuclear detail. Then endocardium and the various valve orifices showed no pathologic change.

The aorta and the medium-sized arteries, particularly the renal and the coronary arteries, were definitely sclerotic and showed thickening of the intima in the form

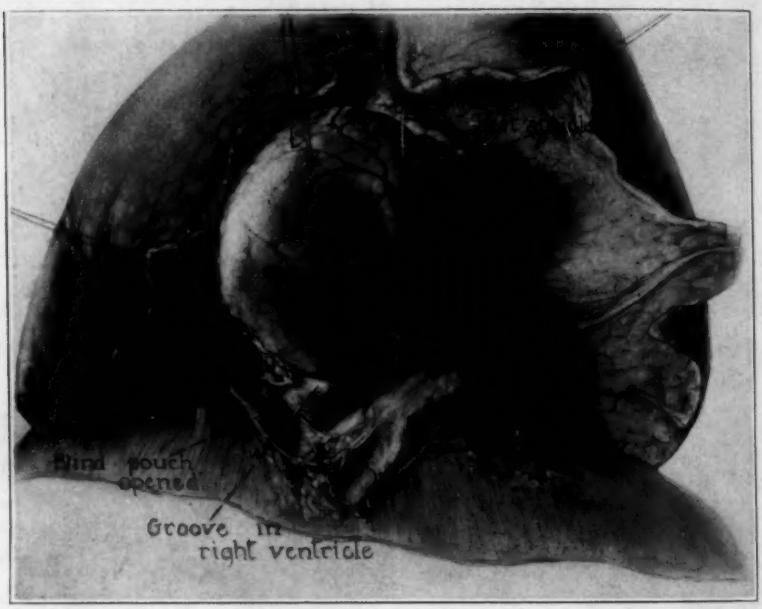


Fig. 2.—The ventricles are elevated, showing the pad of areolar tissue adherent to the atrioventricular junction posteriorly. This pad of tissue extends from the hilus of the left lung to the lateral aspect of the right auricle to which it is adherent. Where it curves forward to its attachment it forms a definite groove in the right ventricle just below the tricuspid orifice.

of elevated pearly gray or yellowish plaques, some of which were calcified. The coronary arteries were widely dilated and showed no evidence of occlusion.

The lung contained a few small areas of consolidation, but in general was air-containing. The right lung was extensively consolidated, and the pleura possessed fibrinous adhesions. The diagnosis of confluent lobular pneumonia was made.

The kidneys together weighed 320 Gm. The capsule was thickened and adherent. The cortex contained a number of stellate scars. Many glomeruli were fibrosed, and the tubules were compressed and atrophic. The vessels were sclerotic. An aberrant renal artery was present on the left side, producing a partial obstruction to the left ureter. The pelvis of this kidney was dilated and contained two calculi.

There was enlargement of the median lobe of the prostate with dilatation and trabeculation of the bladder. The remaining observations were not especially noteworthy.

The case reported was that of an adult who lived fifty-six years, and who had done hard manual labor without any cardiac symptoms. Arteriosclerosis and hypertension developed. A pulmonary infection was followed by cardiac decompensation and death. At necropsy the pericardium was absent. There was some enlargement of the heart. Both ventricles were free, but the right auricle was adherent to several rudimentary bands or folds of pericardium. The latter were such that they did not seem to impair the movements of the heart, and its hypertrophy undoubtedly was that associated with hypertension and arteriosclerosis.

THE FUNCTION OF THE PERICARDIUM

Most textbooks of physiology contain little, if anything, concerning the function of the pericardium. Is this fibrous sac, lined with mesothelium and containing a small quantity of fluid, essential or even useful for the normal function of the heart? Can it sustain a dilating myocardium or is its presence a potential danger, another of those structures like the tonsils, the appendix or the saphenous veins that well could be omitted from the human anatomy?

In an analysis by Moore [1] of forty-two cases of deficient pericardium, adhesions between the heart and adjacent structures were found in twelve, three of which showed enlargement of the heart. In one of these three cases, reported by Bristowe,[3] a valvular defect may have accounted for the hypertrophy. The second case, reported by Curling,[4] occurred in a man, aged 46, who previously had been healthy, who died of myelitis and whose heart was found to be rather large and flabby. The third case, reported by Picchi,[5] was that of a man, aged 60, who died of fibrinous pneumonia; the heart was described as a little larger than normal in volume. Unfortunately, many of the reports of cases of deficiency of the pericardium are incomplete, and an exact analysis of all cases cannot be made. In the case recently reported by Grant [2] there was a small adhesion between the heart and the lung, but this adhesion apparently had no effect on the heart. Likewise in the present case there were several adhesions to the right auricle and along the atrioventricular groove posteriorly, but these adhesions apparently had no effect on the heart. It may be said, therefore, that adhesions between

3. Bristowe: Malformation of the Pericardium and Left Pleura, Tr. Path. Soc. London **6**:109, 1854-1855.

4. Curling, T. B.: Case of Congenital Absence of the Pericardium with Observations, Tr. Med.-Chir. Soc. London **22**:222, 1839.

5. Picchi: Sperimentale **61**:375, 1907.

the heart and adjacent structures occur more frequently when the pericardium is defective, but that there was no conclusive evidence in any of the cases that adhesions were the cause of cardiac hypertrophy.

The cases may be analyzed further to determine whether the heart was more prone to undergo either acute dilatation or a slowly developing dilatation when the "sustaining effect" of the pericardium was not present. In an analysis of forty-six cases by Grant,[2] the size of the heart was not stated in sixteen; ten were in fetuses or children, and of the remaining twenty persons not one died of acute dilatation. Of these twenty cases in adults, the heart was described as normal in ten and as enlarged in ten. Of the ten cases showing enlargement one was associated with syphilis, one with mitral stenosis and one with nephritis; the remaining seven were incompletely reported, so that other possible conditions associated with cardiac enlargement could not be excluded. Among the ten persons with a normal-sized heart, there was no evidence of any cardiac abnormality either in clinical examination or in the history. No case of deficient pericardium was diagnosed clinically, and in no instance was the anomaly of the pericardium regarded as responsible for death or even as having exerted a significant influence.

Experimentally, Barnard[6] found that the heart without its pericardium ruptured under a pressure of one atmosphere, whereas the pressure required to rupture the heart with the pericardium intact was one and three-fourths atmospheres. From these experiments he concluded that the pericardium was necessary to prevent dilatation of the heart. Kuno,[7] in experiments with heart-lung preparations, found that the heart without the pericardium could not tolerate a dilating force, and that a slightly increased venous pressure caused hemorrhage in the cardiac muscle. Amerio,[8] in experiments on rabbits, found that the margin of the pericardium remaining after partial resection in places became adherent to the wall of the chest, but that after total resection of the pericardium no adhesions were found between the heart and the surrounding organs. These procedures, he found, did not affect the life of the animal, and he concluded that in human beings the pericardium, when involved by tumors of the chest wall, could be resected without fear of endangering life. Parlavecchio[9] performed similar experiments on ten dogs, and noted in each case a pronounced loss of

6. Barnard, H. L.: The Functions of the Pericardium, J. Physiol. **22**:43, 1898.

7. Kuno, Yas: The Significance of the Pericardium, J. Physiol. **50**:1, 1915-1916.

8. Amerio, V.: Contributo clinico-sperimentale allo studio delle ferite del pericardio, Atti d. XI Cong. med. internaz., Roma **4**:301, 1894.

9. Parlavecchio: Experimentelle Perikardiektomie und ihre möglichen therapeutischen Anwendungen, Deutsche Ztschr. f. Chir. **98**:126, 1909.

weight and observed at necropsy a hypertrophy of the left ventricle. The size of the heart, however, was not observed by roentgen studies. Rehn [10] performed total or partial pericardiectomy on five dogs, and in no case did he observe any effect on the health of the dog. No adhesions were found at necropsy, and no histologic changes were found in the cardiac muscle. Mazzone [11] removed the pericardium from seven dogs, and found that the operation had no effect on the animals. Yamada,[12] by making a long incision in the pericardium so as to nullify the effect of the pericardium on the heart, was unable in a series of fourteen dogs to obtain the results of Kuno in heart-lung preparations. During the postoperative period, the dogs were exercised by running. Dilatation of the heart did not occur, and in no case was intramural hemorrhage found. From these experiments, Yamada concluded that the protective influence of the pericardium was not of great significance. In none of the experiments mentioned were the animals subjected to strenuous exercise, and the advisability of studying the capacity of the heart to perform a greatly increased amount of work without its pericardium led Beck and Moore [13] to carry out additional experiments. Dogs were vigorously exercised, but not visibly exhausted, by swimming in a tank. From these experiments it was concluded that the general health of the dog did not seem to be impaired by pericardiectomy, that the response to exercise did not vary appreciably from the normal, and that dilatation or hypertrophy of the heart did not develop. Following pericardiectomy, it was found that the epicardium usually became covered with a delicate film of areolar tissue which did not seem to impair the function of the heart. Not infrequently a small adhesion extended to the incised margin of the pericardium or to the lung, but such adhesions seemed to be of little significance.

In certain pathologic conditions the pericardium exerts a pronounced effect on the circulation. Experiments by Beck and Holman [14] in which acute plethora was produced by the intravenous injection of blood and salt solution showed that the pericardium presented a limiting or constricting action to acute dilatation of the heart. In these experiments the pulse pressure doubled and the heart greatly dilated when the peri-

10. Rehn, L.: Zur experimentellen Pathologie des Herzbeutels, Verhandl. d. deutsch. Gesellsch. f. Chir., 1913, p. 339.

11. Mazzone, F.: Contributo sperimentale alla pericardiectomia, Centralbl. f. Chir. **39**:1046, 1912.

12. Yamada, M.: Ueber die Bedeutung des Perikardiums, Mitt. a. d. med. Fakult. d. k. Univ. zu Tokyo **16**:527, 1917.

13. Beck, C. S., and Moore, R. L.: The Significance of the Pericardium in Relation to the Surgery of the Heart, Arch. Surg. **11**:550 (Oct.) 1925.

14. Beck, C. S., and Holman, E.: The Physiological Response of the Circulatory System to Experimental Alterations: II. The Effect of Variations in Total Blood Volume, J. Exper. Med. **43**:681 (Nov.) 1925.

cardium was opened. Numerous dropped beats and extrasystoles marked the opening of the pericardium and the subsequent dilatation of the cardiac chambers. Whether the pericardium prevents stretching of a diseased muscle fiber to the point at which it no longer can effectively contract is problematic. It is to be noted, however, that in the experiments on acute plethora the cardiac output greatly increased when the restraining influence of the pericardium was removed. According to the experiments recently reported by Van Liere and Crisler,[15] vagal stimulation resulted in a greater dilatation of the heart after the pericardium had been removed than occurred with the pericardium intact.

In the mechanism of cardiac tamponade the pericardium plays a deleterious rôle on the circulation. Under the influence of a gradually increasing tension the pericardium enlarges to contain either the dilating heart or an increased collection of fluid. As fluid collects in the pericardial cavity the heart decreases in size, the venous pressure rises and the arterial pressure falls. The formation of fluid must take place slowly, so that the pericardium can dilate sufficiently to contain it; otherwise a fatal tamponade results. In no event can the intrapericardial pressure rise above the pressure in the venae cavae.

In adhesive pericarditis the pericardium likewise plays a deleterious rôle. In those cases in which the pericardium is thickened and shrunken, the heart inadequately fills with blood, and its contractility is impaired much as a tight-fitting glove impairs the free movements of the hand. It is in these cases that pericardiectomy gives highly beneficial results.

SUMMARY AND COMMENT

A case of deficiency of the pericardium is described.

An analysis of the cases that have been reported shows that adhesions between the heart and adjacent structures occur more frequently with this anomaly than when the pericardium is present. In no instance was the anomaly of the pericardium regarded as responsible for death or even as having exerted any significant influence on the heart or the circulation. The potential danger of bands of tissue adherent to the heart, however, should not be overlooked.

There is no evidence to indicate that the pericardium exerts a beneficial effect on the acutely dilating heart.

In such pathologic conditions in which a positive intrapericardial pressure occurs or in which cardiopericardial adhesions develop, the presence of the pericardium may constitute a grave danger to the mechanics of the circulation. In the therapy for these conditions surgery has much to offer.

---

15. Van Liere, E. J., and Crisler, George: The Influence of the Pericardium on Acute Cardiac Dilatation Produced by Vagal Stimulation, read at the meeting of the American Physiological Society, Chicago, March 27, 1930.

THORACOPLASTY AS A METHOD OF TREATMENT IN PULMONARY TUBERCULOSIS

REPORT OF FIFTY-THREE CASES *

B. N. CARTER, M.D.
CINCINNATI

Surgical collapse of the thoracic wall as a method of treatment for selected cases of pulmonary tuberculosis has come to be regarded as a valuable addition to the usual measures previously employed in combating this disease. During the past five years I have performed fifty-three thoracoplasties. Sufficient time has elapsed in many of these cases to enable one to judge the effectiveness of the operation. In addition, it has been possible to follow all of the cases so closely as to be able to state with accuracy the end-results. Forty-six of these fifty-three cases have been referred to the Surgical Service of the Cincinnati General Hospital by the Hamilton County Tuberculosis Sanitarium.

INDICATIONS FOR OPERATION

The need for close cooperation between the surgeon and internist in dealing with the surgical treatment of patients with tuberculosis is obvious. The end-results of thoracoplasty depend largely on the proper selection of patients, and the surgeon who is so fortunate as to have the advice of a careful and well-trained internist is almost sure to show a high percentage of cures. Thus the figures of the Sauerbruch Clinic [1] show that of thirty-five patients with strong indications and no contraindications, 40 per cent were cured, 51 per cent improved and 6 per cent died; whereas, of forty-three patients with questionable indications and some contraindications, only 14 per cent were cured, 60 per cent improved and 14 per cent died. The commonly accepted indications for thoracoplasty are a chronic fibrous type of tuberculosis with or without a cavity, essentially unilateral, in a patient who has shown evidence of good resistance, in whom treatment in a sanitarium has failed to effect a cure and in whom artificial pneumothorax cannot be performed. The inflexible indications to my mind should be: (1) evidence of resistance as indicated by fibrous tissue reaction around the lesion, with contraction of the wall of the chest and dislocation of viscera; (2) the

* Submitted for publication, May 2, 1930.

* From the Department of Surgery, University of Cleveland.

1. Bruner, A., and Baer, G.: Die chirurgische Behandlung der Lungentuberkulose, Berlin, Julius Springer, 1925.

impossibility of performance of a satisfactory pneumothorax. As a general rule, the rapidly progressing caseous types of tuberculosis with little or no formation of fibrous tissue do not seem suitable for operation, although good results have been reported by Hedblom in one such case in which preliminary phrenicotomy was performed, followed by complete thoracoplasty in many stages over a period of seven months. In my series, the average duration of tuberculosis before operation was three years, the shortest time elapsing from recognition of the disease until operation being one year, and the longest ten years.

Claus' epigram, "A good pneumothorax is better than a thoracoplasty, but a good thoracoplasty is better than a poor pneumothorax," about sums up the attitude of most observers at the present time. Certainly, the majority of the members of the medical profession feel that if a satisfactory pneumothorax can be done, a thoracoplasty is not indicated. The reasons for this, briefly, are that pneumothorax is a simple procedure, quickly and easily done; it allows gradual compression of the lung, can be terminated when desired and gives the most complete compression of the lung; in contrast, a thoracoplasty is a major surgical procedure; it gives sudden compression of the lung; it cannot be abolished and does not give the most complete compression. The advantage of thoracoplasty over pneumothorax is that once done, thoracoplasty is permanent, whereas with pneumothorax, refills with their accompanying dangers and inconveniences have to be carried out over long periods of time. Pneumothorax is associated with certain dangers such as empyema, rupture of the lung, spontaneous pneumothorax on the opposite side and pleural shock, but thoracoplasty also may be complicated by the even graver dangers of shock, infection and aspiration pneumonia. It is not so much a question as to whether or not pneumothorax is preferable to thoracoplasty, for that seems established, but as to when to substitute thoracoplasty for pneumothorax. In this connection, the results of Matson [2] and his co-workers are of interest. In a series of 492 cases in which pneumothorax was attempted the following results were obtained: (1) complete collapse in 43 per cent, or 211 cases, with clinical recovery in 48 per cent; the condition arrested in 20 per cent and death in 21 per cent; (2) partial collapse in 37 per cent, or 183 cases, with clinical recovery in 13 per cent; the condition arrested in 13 per cent and death in 50 per cent; (3) no collapse obtainable in 20 per cent, or 98 cases, with recovery in 7 per cent; the condition arrested in 23 per cent and death in 55 per cent. Swezey and Schonbar,[3] in reporting on 207 cases (205 of tuberculosis) in which pneumothorax was done, over a period of ten years, found that successful collapse could

2. Matson, R. C.; Matson, R. W., and Bisaillon, M.: Tubercle **7**:12, 1925.
3. Swezey, S., and Schonbar, Alexander: Am. Rev. Tuberc. **13**:156, 1926.

be obtained in 38 per cent, partial collapse in 36 per cent, and that in 26 per cent collapse could not be obtained; their mortality figures are much like those of Matson and his co-workers. Mattill and Wall [4] stated that of 150 patients on whom pneumothorax was attempted, no free pleural space was found in 37; of these, 19 were subjected to thoracoplasty, with the result that 4 died, 4 are working and 11 are under treatment; of the 18 who were not operated on, 7 died, 1 is working and 10 are under treatment. Judging from these reports, which are probably representative of the usual experience, pneumothorax can be effectively employed in only about 40 per cent of the cases, and whereas the percentage of deaths is approximately 20, if pneumothorax is complete, it is about 50 when pneumothorax is partial or impossible. It would seem, therefore, that in a case suitable for collapse therapy, but in which this may not be thoroughly accomplished by pneumothorax, it is far safer to do a thoracoplasty, and this should be done early, and not as a last resort. The instances in which thoracoplasty is preferable to pneumothorax have been well stated by Davies [5] as follows:

> (1) In the long standing chronic cirrhotic type of pulmonary tuberculosis which is associated with great shrinkage of the lung and consequently displacement of the mediastinum and with considerable and generalized dilatation of the bronchial tree. (2) In cases with large thin walled or very thick walled cavities. In the former the danger of rupture in the pneumothorax is great. In the latter it is very improbable that the pneumothorax can produce an efficient collapse.

The same author stated that thoracoplasty should replace pneumothorax:

> (1) When adhesions are present which are interfering with the efficient collapse of the lung. The exception to this is when the adhesions are basal only. (2) Where there is a thin walled cavity held open by an adhesion or adhesions too broad for division by cautherization with safety. (3) When there is a thick walled cavity which cannot be collapsed by reason of the rigidity of its walls. (4) When there is an effusion which is persistent and is responsible for, part at any rate, of the febrile condition. (5) When there is a bronchial fistula. (6) When the lung is creeping out or has crept out and the symptoms are recurring, and the reexpansion is too great to be dealt with by phrenic evulsion only.

Six of my patients had had a pneumothorax (artificially induced) which was not satisfactory owing to the accumulation of thick, tuberculous pus in two cases, to an uncollapsed cavity in two, and to a combination of each of these in two. They have all done unusually well and are now classed as apparently well. They stood operation better than the usual patient. The degree of collapse of the thoracic wall was excellent, and the pleural cavity has been well obliterated in all.

4. Mattill, P. W., and Wall, M. H.: Am. Rev. Tuberc. **13**:537, 1926.
5. Davies, H. M.: Tubercle **7**:437, 1925.

The indications for thoracoplasty admitting of variations are the condition of the opposite lung and the general condition of the patient. In the all important question of the good lung, the internist who has been familiar with its appearance in the roentgenogram and the physical signs over a period of months, can and should bear the burden of the decision. There are very few cases of absolutely unilateral pulmonary tuberculosis, and nearly all cases coming to operation show some degree of involvement on the opposite side. Lesions on the good side which seem of serious nature to the surgeon seeing the patient for the first time, in the light of the internist's knowledge, may not be contraindications to operation. The latter may know that such lesions have been stationary for weeks or months, or that they are becoming more fibrous or that they may be due to circulatory changes. Active caseous lesions on the good side are definite contraindications to operation. Inactive, fibrotic lesions or less fibrotic ones that have been stationery are not contraindications. Other things being equal, lesions situated in the apex of the good lung are less serious contraindications than similar sized ones in the base or hilus. The explanation usually given for this is that the excursions of the lung are greater at the base than at the apex, and that the hilus, being in proximity to the heart, is kept in almost constant motion; consequently, lesions in these locations are more apt to be activated when increased work devolves on the good side, following collapse of the bad.

Changes occur so quickly in the more affected side, that no patient should be operated on unless careful physical and roentgen examinations are done just before the operation. In one of my cases, in which this was neglected, I have reason to believe that changes contraindicating operation had occurred within ten days following the last roentgen examination and were unrecognized, with disastrous results. In another, a roentgenogram made two weeks before the proposed operation showed the good side clear, but one made the day before that set for operation, showed contraindicating changes. The operation was deferred for six months, the lesions receded or became fibrosed and operation was successfully done.

The desire to extend the indications for operation is unusually strong in the case of these patients whose only hope lies in surgical collapse of the lung, and physicians are tempted to take desperate chances. In doing so, I feel that it simply makes the patient's end more painful and tends to discredit the operation. By means of operations divided into more than the usual two stages and preceded by phrenicotomy, good results may be obtained in cases that would otherwise be fatal. I feel that one of my patients who died could have been saved, had she had a phrenicotomy first, and then a several stage operation rather than the

accustomed two stages. By closest attention to detail before operation, patients who at first glance seem unable to stand the ordeal, may be gotten into satisfactory condition for it. The greatest aid here is the use of transfusion. This has been emphasized by Yates,[6] and we heartily agree with him as to its use both preoperatively and postoperatively. I have used it in nine cases in this series; three transfusions were given in two of these, one before each stage of the operation. In every case but one, the results of transfusion were remarkable.

Attention to several details in the preoperative treatment will be followed by greater ease of operation, fewer complications and better results. It is advisable to have the patients under observation for several days in the hospital where the operation is to be performed. The excitement of being transported to a new hospital, the fatigue or exposure on the journey may cause a flare-up of temperature and pulse, in the presence of which the added strain of operation might be dangerous. Practically always, these patients, ill and toxic for a long time, have a myocarditis, and a treatment with digitalis should be given before operation. This precaution was disregarded in some of my first cases, and the contrast in their cardiac reaction was striking in comparison to the later ones in which digitalis was used. Patients with appreciable amounts of sputum should be made to empty their cavities by postural drainage just before operation, to guard against aspiration during or immediately after the collapse of the thoracic wall. It is imperative that the skin be free of abrasions, acne, etc., before operation is attempted. Probably the most important precaution preoperatively is to match the patient's blood for transfusion. So frequently, the long illness has depleted the patient's reserve and has brought about such an anemia, that they are especially poor risks. It is remarkable what a difference one or two transfusions will make in the patient's condition, and I am sure that by using them freely, certain patients who would otherwise succumb, can be gotten safely through the operation.

ANESTHESIA

The tendency at present is toward general anesthesia for cases of thoracoplasty, but nevertheless, we favor and use local anesthesia. Archibald [7] has given up the use of local anesthesia for a combination of gas and oxygen and local anesthesia. Sauerbruch [8] uses ether in patients whose expectoration of sputum in twenty-four hours is less

6. Yates, J. L.: Rationale of Operations Helpful in Promoting Recoveries from Pulmonary Tuberculosis, Arch. Surg. **14**:369 (Jan.) 1927.

7. Archibald, E.: Am. J. Surg. **10**:328, 1925.

8. Sauerbruch, F.: Die Chirurgie der Brustorgane, ed. 2, Berlin, 1920, vol. 1.

than 30 cc. Chalier [9] stated that he used rectal anesthesia. Meyer [10] seems to feel that local anesthesia is safest. General anesthesia is certainly open to the serious objection of aspiration of the contents of cavities, so frequently present, into other portions of the same or opposite lung. This constitutes the greatest danger during the operation, and we feel that any procedure that will tend to decrease its incidence is well worth while. One of my patients died of pneumonia in the good lung, which I felt was the result of a light gas and oxygen anesthesia. The objections usually made to the local anesthesia are that it is frequently unsatisfactory, it is time-consuming, and poisoning from procaine hydrochloride with death may result, and that it is too great a mental strain on the patient and surgeon. I have used local anesthesia in fifty of the fifty-three cases, and still feel it is the method of choice. A combination of light nitrous oxide gas and local anesthesia was used in three cases. The method used has been direct infiltration of the skin and muscles and injection of the intercostal spaces close to the spine under direct vision. The intercostal nerves are injected as soon as the spaces are exposed and by the time a few vessels have been tied off and the soft tissues reflected laterally, the anesthesia is complete. I have had one case in which local anesthesia was unsatisfactory and had to be abandoned in favor of light gas and oxygen. The infiltration can be done rapidly; the usual time required in my cases in which the anesthesia is used, is about fifteen minutes. There have been no cases of serious poisoning with procaine hydrochloride, although in one instance it was felt wise to terminate the operation after resecting three ribs owing to a reaction attributed to the procaine hydrochloride. One per cent procaine hydrochloride is used for the skin; 0.5 per cent for the soft tissues and 1 per cent for the intercostal nerves. Matson [11] has shown that in injecting solutions into the intercostal spaces close to the spine, not infrequently some of the solution is found inside the dura. He expressed the belief that this may be the cause for some of the deaths and reactions attributed to procaine hydrochloride. It is quite true that local anesthesia imposes a more severe mental strain on the patient, and this is its worst feature. However, by preliminary administrations of morphine and by encouragement from a cheerful and tactful nurse during operation, a good deal may be done to lessen it. Although it is more trouble and is harder on the operator, the fact that it is safer should overbalance these more or less minor objections to its use.

---

9. Bonniot: Presse méd. **32**:1012 (Dec. 17) 1924.

10. Meyer, Willy: Anesthesia in Extrapleural Thoracoplasty for Pulmonary Tuberculosis. Arch. Surg. **14**:432 (Jan.) 1927.

11. Matson, R. C.; Bisaillon, M., and Matson, R. W.: Tubercle **7**:65, 1925.

OPERATION

The standard Wilms-Sauerbruch thoracoplasty is so well known at present that it is not necessary to go into its details. This is the procedure used by the majority of surgeons today who are operating on patients with pulmonary tuberculosis. Although there are many modifications, they are for the most part minor departures from the standard operation, such as variation in the length of the rib resected; which rib is divided first and whether the lower or upper half of the chest is attacked first. Thus Brauer [12] resects greater lengths of rib beneath the scapula than other operators. Bull [13] does an apicolysis beneath the three upper ribs before dividing them. John Alexander [14] recommends phrenicotomy followed by resection of only the upper seven ribs, etc. There seems to be a unanimity of opinion on the following points: (1) the division of the ribs close to their articulation with the spine, (2) resection of a portion of the first rib, (3) resection of longer portions of the ribs than Wilms advocated. Archibald's [7] method of fitting the amount of rib resected to the condition in the individual case has seemed most logical to me and that has been the method used in this series. In general, however, I resect 10 cm. of the eleventh rib; from 12 to 15 cm. of the tenth to the fourth ribs inclusive; 10 cm. of the third rib; 8 cm. of the second rib, and from 3 to 5 cm. of the first rib. Of the 53 patients in this series 26 had segments of the first to the tenth ribs inclusive resected; 19 had portions of the first to the eleventh ribs inclusive, 2 had the first to the twelfth ribs inclusive, and 3 had the first to the ninth ribs inclusive. In the first few cases I made a curved incision out under the scapula for the second stage, but have abandoned it and now prolong the lower end of the second incision into the upper end of the first. My average for the total length of rib resected has been 115 cm. for 11 ribs and 125 cm. for 11 ribs. I always resect the first rib and usually the eleventh, though if the diaphragm is high, if a preliminary phrenicotomy has been done, or the lower lobes are relatively free from disease, I leave the eleventh rib intact. Owing to the fact that the contraction of the fibrous tissue in the lung on the side on which the operation has been performed is the most potent factor in producing the collapse, following resection of the ribs, greater lengths of rib should be resected in cases in which there is relatively much scar tissue in the diseased lung. It is interesting to note the varying amount of collapse in the different portions of the same lung, the collapse being more marked over the portions containing the greatest amount of fibrous tissue. Where there is little fibrous tissue in the lung and pleura and

12. Brauer, L.: Beitr. z. Klin. d. Tuberk. **51**:319, 1922.
13. Bull, P.: Proc. Roy. Soc. Med. **17**:1, 1924.
14. Alexander, John: Ann. Surg. **81**:748, 1925.

consequently, greater mobility of the mediastinum, the danger of flapping mediastinum with its resulting circulatory and respiratory embarrassment should be guarded against by resecting shorter segments of rib, and by dividing the operation into more than the accustomed two stages. I have advocated removal of portions of the first to the tenth ribs or the first to the eleventh ribs whenever a thoracoplasty has been advised and partial thoracoplasties have not been done. Though I have not had experience with the latter, I have felt that the amount of lung conserved by it is not comparable with the greater degree of collapse obtained by the resection of portions of all the ribs. All of my patients have had the portions of the lower ribs resected first, followed in about two weeks by resection of portions of the upper ones. Except for six cases, preliminary phrenicotomy has not been done. However, now I believe it advisable in nearly all cases, and especially those in which (1) it is uncertain whether the good lung will stand the added strain thrown on it by thoracoplasty; (2) the patients general condition is such that it is questionable whether he can stand thoracoplasty in his present state, phrenicotomy being done to lessen fever, improve the general condition, and (3) in which it is possible that phrenicotomy alone may effect a cure.

The operative procedure used in this series of cases varies in several respects from those reported from other clinics. One gets the impression from the literature that the operations must be done with extreme rapidity, as though the time element was a great factor in their success. It has been my opinion that an operation done only as rapidly as is compatible with gentle handling of tissues, with accurate hemostasis and absence of tearing and tugging, is far safer, is attended with less shock, and more quickly recovered from, than one done under conditions where much has to be sacrified to the demands of speed. The average time in my cases is about an hour. The patients are placed in a supine position on the operating table, with the arm on the affected side hanging over the edge so it can be moved to throw the scapula out from the thorax. As soon as the skin is incised, towels are fastened to the edges of the wound with small metal skin clips. This can be done rapidly and prevents slipping better than any method we have tried. Gentleness in handling tissues and accurate hemostasis are insisted on. Fine silk has been used for ligatures and suture in all but one case and that wound became infected. No case has been drained and only three have filled with blood or serum to a degree necessitating aspiration. All the operations have been done in two stages, from below up, save five, which were done in three stages. Two of these were in emaciated patients who expectorated from 4 to 6 ounces of sputum a day, had tuberculous laryngitis and were in poor general condition. One other was in an

emaciated woman, aged 42, whose condition became alarming after three ribs had been resected, so that the operation was terminated and two later stages done. In the other instances, the presence of dangerous lesions on the good side made a three-stage resection preferable so as to more gradually throw added strain on the good lung. Preliminary phrenicotomy has been used only six times, although I now believe it should have been done in many of the other cases. The usual interval between operations has been two weeks; in some cases, for various reasons, the operation has been done with intervals of 10 days, and in others intervals of over two weeks. If there is any doubt as to the patient's ability to stand the second operation, it is wiser to wait longer than to regret a too hasty second stage.

No serious accidents have been met with during operation. The pleura was torn in two cases but was immediately sutured and reinforced with intercostal muscle. No ill effects resulted, and the wounds healed without complications. One patient had a severe reaction, which I thought due to procaine hydrochloride, as mentioned before. The first rib was very difficult to expose in several cases. I have not been troubled with alarming degrees of shock either during or after operation in any case, although several patients have exhibited mild degrees as evidenced by pallor, slight sweating and rapid pulse. It is remarkable how well the patients stand this major procedure if properly prepared, and if the operation is done without loss of blood and without undue traumatism to tissues.

POSTOPERATIVE CARE

The postoperative management resolves itself largely into preventing aspiration pneumonia and into maintaining the resistance of the patient. The first forty-eight hours are the most trying and dangerous, for it is then that aspiration is most apt to occur, owing to the difficult and painful attempts at coughing. The frequency of the cough should be controlled by morphine, which also relieves the pain to such degree that when coughing takes place it can be effective. Every effort is made to have the patient expectorate the secretions when he begins to cough. Firm pressure on the operated side during coughing spells and a very snug dressing help greatly to accomplish this.

The general resistance is maintained by forcing fluids, by partaking of nourishing food, by rest (sedatives) and by transfusion, if other methods fail. I believe that many lives will be saved by transfusions made before and after the operation. Mediastinal flutter and paradoxic respiration are the most serious sequelae of the operation. They are apt to occur in patients with a thin pleura. Consequently, in this type of case where, as aforementioned, shorter portions of ribs should be resected, it is safer to do the operation in three stages. In this series

one patient died from these causes on the second day after the operation. She was extremely well nourished, in good general condition and had a small cavity at one apex with the rest of the lung relatively clear. It was decided to resect the upper eight ribs at one operation. The pleura was so thin that the lung could be seen moving beneath. The patient stood the operative procedure well but in a few hours developed a rapid pulse cyanosis, pallor and dyspnea. In spite of all that could be done, she died in thirty-six hours.

The temperature of patients who do well is usually elevated from one to three degrees for the first two days, then falls steadily, reaching the preoperative level in about one week. This is thought to be due to a flooding of the body with toxins from the collapsed lung; it may be likened to a large injection of tuberculin. Bull [13] and Graf [15] have found that blood taken from patients in the febrile stage after thoracoplasty is much more toxic to tuberculous guinea-pigs than that of the same patient when taken before operation. When the temperature remains high, or becomes more elevated, the explanation usually lies in infection of the wound or extension of the disease to other portions of the lung on the same side, or to the opposite side by aspiration; this was true in both of our cases in which aspiration pneumonia developed on the opposite side. There is much less reaction following the stage in which the upper ribs are resected. If the patient has stood the resection of the lower ribs, I feel no alarm as to his ability to go through the second stage. It is largely for this reason that we resect the lower ribs first; for as it is the greater strain of the two, the patient has a better chance of standing it when his resistance is at its highest, i. e., before there has been any operative procedure. The pulse in general follows the temperature. Where digitalis has been used before and after operation I have found much less cardiac reaction. Nearly every case shows slight dyspnea or cyanosis for a few days after operation. Marked cyanosis and dyspnea mean usually a flapping mediastinum, but may result from aspiration pneumonia in the good lung. I have seen it three times in my cases. In two instances there was tuberculous pneumonia in the good lung and in the other there was mediastinal flutter. The dressings are undisturbed for five days, at which time the sutures are removed and a tight dressing applied for another week.

RESULTS

Fifty-three patients have been operated on in the past six years, a total of 108 operations (45 cases in two stages, 5 cases in three stages and 3 cases with only one stage completed). Forty-four patients

15. Graf, W.: Deutsche Ztschr. f. Chir. **187**:418, 1924.

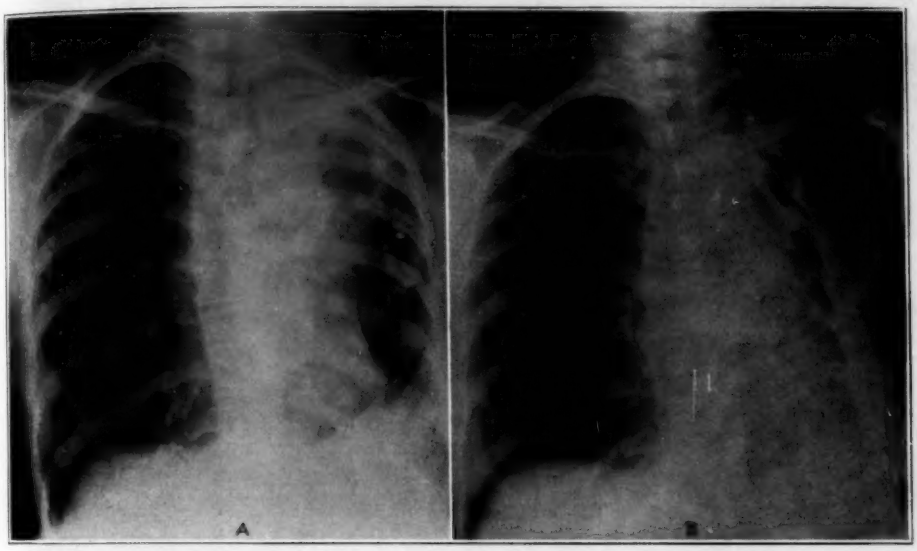


Fig. 1.—H. H. Roentgenograms showing (*A*) large cavity at the apex of the left lung; (*B*) result one year after thoracoplasty.

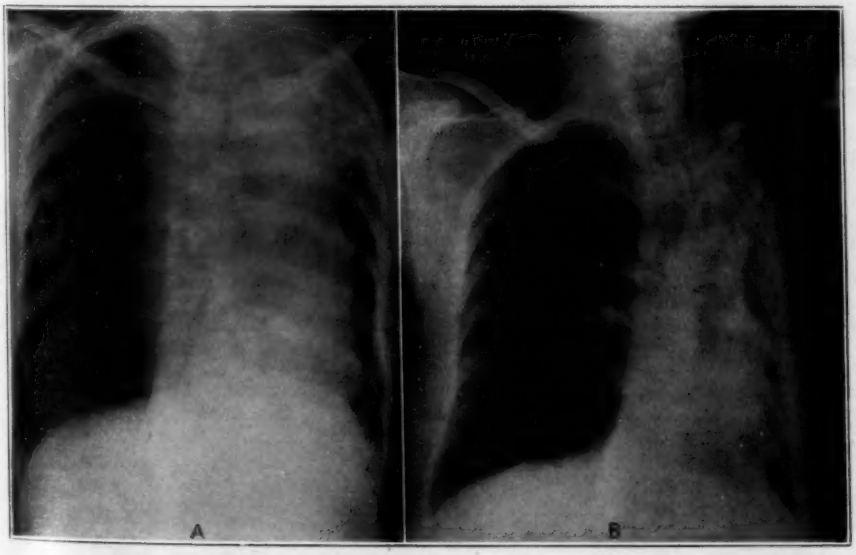


Fig. 2.—M. J. Roentgenograms showing (*A*) multiple cavities and considerable fibrosis on the left; (*B*) results two years after operation.

(83.01 per cent) are alive today; 9 (16.99 per cent) have died. Table 1 gives the results to date in the 53 cases. In this table "apparently well" indicates that the patient has a sputum that is negative for tuberculosis

TABLE 1.—*Results in Fifty-Three Cases*

Year in Which Operation Was Performed	Number of Cases Done	Apparently Well	Condition of Patient at Present			
			Improved	Unimproved	Too Early to Classify	Dead
1924	1	..	..	..	..	1
1925	5	3	..	..	..	2
1926	13	9	1*	1	..	2
1927	11	9	..	..	..	2
1928	9	6	3†	..	..	0
1929	14	8	1‡	..	3§	2
Total	53	35	5	1	3	9

* This patient, a girl, was 16 years of age when she was operated on four years ago. She was a desperate risk, thin and anemic with a huge cavity occupying nearly the whole upper lobe. The cavity has been obliterated, the sputum was negative for tuberculosis bacilli on discharge, and the patient was apparently well for two years, but has recently been feeling tired, has been coughing more and was put to bed two months ago. I believe she will recover, and if she is careful, live comfortably for years.

† One of these three patients had a huge cavity in one apex which, according to a roentgenogram, has disappeared, though actually I believe it is present, but small. The patient looks healthy, feels well and feels that he is cured. Except for an occasional positive sign of tuberculosis bacilli in the sputum, he could be classed as "apparently cured." Another is a woman, aged 40, with a very thick-walled cavity which has not been entirely collapsed. She looks and feels well but still has a few tuberculosis bacilli in her sputum. The last patient of the three is a boy who was 18 when the operation was done. He was a poor risk and had his operation in three stages, with several transfusions between. His lung showed several cavities in upper and middle lobes. He looks and feels much better, has a moderate cough, but his sputum shows a few bacilli.

‡ This patient is a man, aged 38, who has been sick for six years. The whole upper lobe on the left was a huge cavity. The cavity has been decreased to about one-tenth its previous size but is still present. The patient shows positive signs of tuberculosis bacilli in the sputum. Further operative procedure is to be done in an effort to collapse the cavity completely.

§ These three patients all have sputum that is negative for tuberculosis bacilli and seem on their way to recovery, but it is too early to say what their end-result will be, as they have been operated on only three months ago.

TABLE 2.—*Summary of Ages, General Condition and Sex of Patients and Side of Operation*

Recovered												
Age						General Condition						
15 to 20	21 to 25	26 to 30	31 to 35	36 to 40	41 to 50	Good Risk	Moderate Risk	Poor Risk	Male	Female	Right	Left
4	15	6	9	5	5	30	9	5	15	29	19	25

Died												
Age						General Condition						
21 to 25	31 to 35	36 to 40	41 to 50	51 to 60		Good Risk	Moderate Risk	Poor Risk	Male	Female	Right	Left
5	1	2	1	1		3	2	4	4	5	4	5

bacilli is free from cough, and is leading a normal life; "improved" means that the patient has sputum that is occasionally positive for tuberculosis bacilli, has gained in weight and strength and is able to be up and about but has to take unusually good care of himself.

In table 2 is found a summary of the ages of the patients operated on, their general condition, sex and side of operation.

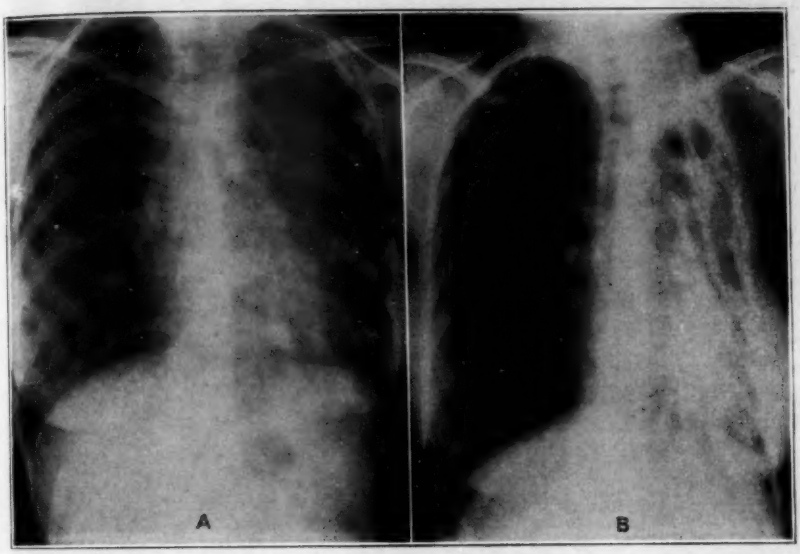


Fig. 3.—H. L. Roentgenograms showing (*A*) huge cavity at the apex of the left lung; (*B*) result eighteen months after thoracoplasty.

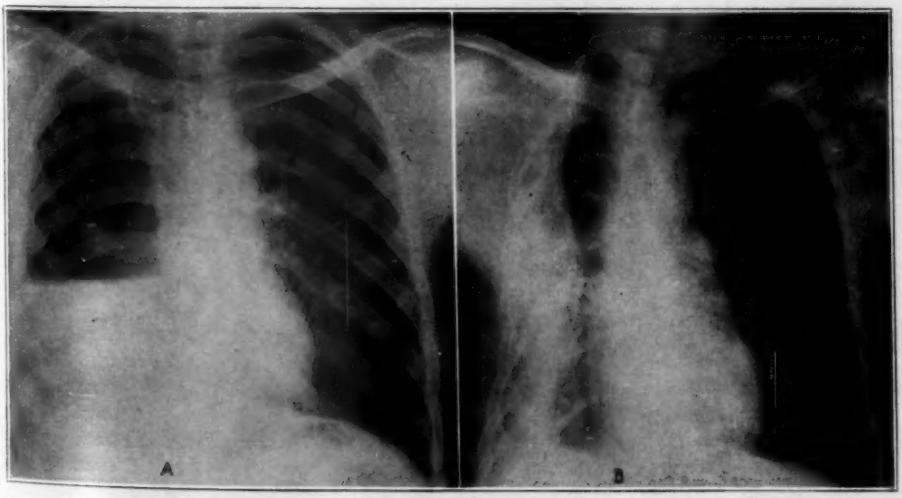


Fig. 4.—W. L. Roentgenograms showing (*A*) artificial pneumothorax with repeated accumulation of thick fluid showing many tubercle bacilli; (*B*) result one year after thoracoplasty.

It is our plan to deal with the pathologic condition of the "good" and the "affected" lungs in a separate communication which will be published shortly. Table 3 gives a very crude idea of the condition of the affected lung in this series.

There was evidence of extrapulmonary tuberculosis before operation in seven of the fifty-three patients. Three of these patients have died. Their lesions consisted of laryngitis, cervical adenitis and gastro-intestinal and laryngeal tuberculosis in the last. In the four patients who are classed as "apparently well," the extrapulmonary lesions were rectal fistula in two cases, and epididymitis in two.

Nine deaths have occurred in this series; an analysis of each death follows:

Case 1.—S. M., a woman, aged 24, ill for one year with tuberculosis was moderately well nourished. She became slightly cyanotic on little exertion. She had a history of digestive upsets, vomiting and alternating attacks of diarrhea

Table 3.—*Condition of Affected Lung*

	Living	Died
Cavity	7	
Cavity and caseous bronchopneumonia	15	5
Cavity and marked fibrosis throughout the lung	10	4
Cavity, fibrosis and fluid	4	
Fibrosis	1	
Fibrosis, fluid and caseous bronchopneumonia	1	
Artificial pneumothorax unsatisfactory		
(a) Cavity uncollapsed	2	
(b) Fluid thick, purulent with many tuberculosis bacilli	2	
(c) Combination of a and b	2	

and constipation with slight hoarseness at times. Roentgenograms showed a huge cavity at the apex of the left lung with much caseous bronchopneumonia below it. The right lung showed "fans" resembling caseous bronchopneumonia in the first and second interspaces trunks. There was very little fibrosis on either side. Six ounces of sputum were expectorated in twenty-four hours. This was the first case of the series. Thoracoplasty in two stages was done in August, 1924, the huge cavity was not entirely obliterated by operation. The patient improved for two months, then showed increasing gastro-intestinal involvement, and died five months later with evidences of tuberculous involvement of meninges, kidneys and intestines. In the light of our present knowledge, she was a poor subject for the operation owing to her lack of resistance as evidenced by little fibrosis and the rapidity of the progress of the disease, exudative lesions in the good lung and the extrapulmonary involvement.

Case 2.—M. G., a woman, aged 40, had been ill for five years with tuberculosis. She was thin and weak, and became slightly cyanotic and dyspneic on exertion. There was no history suggestive of extrapulmonary tuberculosis. Roentgenograms made three weeks before operation showed a large cavity at the apex of the left lung with considerable fibrosis and dislocation of viscera; the right lung was essentially clear. She expectorated 4 ounces of sputum in twenty-four hours. The temperature was 102 F. daily. Thoracoplasty was done in two stages in November, 1925. A good collapse ensued. Roentgenograms made ten days after

Fig. 5.—Photograph showing the two separate incisions for the two stages of thoracoplasty. This has been discarded for the incision shown in figure 6.

Fig. 6.—Photograph showing the type of incision now used.

the first operation showed tuberculous caseous pneumonia in the lower lobe of the good lung. The patient died three months after operation from extensive involvement of the good lung. I feel that we should have had a roentgenogram immediately before the operation, and not have relied on the one done three weeks before. Had we done so, I think we would have found enough tuberculous involvement of the good lung to have contraindicated operation. Provided there was no involvement of the good lung before operation the case would have been much better handled had we done a preliminary phrenicotomy and a three-stage thoracoplasty with a transfusion between each stage.

Case 3.—G. P., a woman, aged 39, had been ill for one year with tuberculosis. She was thin, but a moderately good risk. She had temperature of 102 F. each afternoon, and a pulse rate of 120. Roentgenograms showed cavities in upper part of the left lung; fibrosis with retraction of ribs and dislocation of the viscera. The good lung showed fibrosing lesions in the second interspace trunk; 3 ounces of sputum was expectorated in twenty-four hours. A preliminary phrenicotomy was done in December, 1925, and one week later the temperature had fallen from 102 to 100 F. each afternoon. A partial thoracoplasty (tenth to sixth ribs, inclusive) was then done under local and gas anesthesia. The patient was so apprehensive that gas had to be used. Following operation, the temperature rose to 103 F. and remained up each day. A roentgenogram showed extensive tuberculous pneumonia on the good side, and the patient became progressively worse, dying in May, 1926, six months after operation. Autopsy showed ulceration in the cecum and ileum and extensive cavitation and caseous bronchopneumonia throughout both lungs. This patient died from involvement of the good lung following operation possibly due to aspiration during the operation while under gas anesthesia.

Case 4.—W. W., a man, aged 53, had been ill with tuberculosis for one year. He was moderately well nourished; had a normal temperature; and tuberculous laryngitis which was healing. Roentgenograms showed a cavity at the upper part of the right lung, with considerable fibrosis and dislocation of viscera. The good lung showed a large area of fibrosis, the site of an old cavity which appeared healed. The patient was operated on in three stages under local anesthesia, and a good collapse of the lung followed. He did well for six months, but died ten months after operation from an extension of the disease to, or a lighting up of the old process, in the good lung.

Case 5.—C. C., a man, aged 23, had been ill for two and one-half years with tuberculosis. Phrenicotomy was done in October, 1925. Improvement followed until July, 1926, when cavitation began to appear in the affected lung and the good lung showed several small areas of caseous bronchopneumonia in the apex. By November, 1926, these lesions in the good lung had become fibrosed. Thoracoplasty in two stages was done in November, 1926, and the patient had an uneventful recovery. Six months after operation tuberculosis of the spine and kidneys developed, and he died of generalized tuberculosis twelve months after operation.

Case 6.—G. P., a man, aged 49, had been ill for nine years; he was emaciated, weak with frequent cough and expectoration of several ounces of sputum a day. He had an active tuberculous laryngitis and a few enlarged cervical glands. Roentgenograms showed marked fibrosis in the right lung with much contraction and a large cavity at the apex. The good lung showed marked fibrosis and calcification; there was no activity. Owing to the marked emaciation and weakness, a three-stage thoracoplasty was done in August, 1927, with a transfusion between each stage. The patient stood the operative procedure well. The laryn-

gitis improved for several months then flared up, and the patient succumbed nine months after operation with evidences of a generalized tuberculosis.

CASE 7.—M. P., a woman, aged 21, was very well nourished and in excellent condition. She had been ill with tuberculosis for two years, and under sanitarium regimen had improved remarkably. All that remained of an extensive tuberculosis was a small thick-walled cavity at the apex of the right lung. The remainder of the lung fields had cleared up entirely. A thoracoplasty involving the first to the seventh ribs was done under local and gas anesthesia in one stage. The pleura was very thin, and the lung could be seen moving to and fro beneath it. The patient's color was bad at the close of the operation, but the pulse was good for twenty-four hours. Typical paradoxic respiration and mediastinal flutter developed, and she died forty-eight hours after operation. I feel now that when it was seen how thin the pleura was, shorter segments of rib should have been removed and only three or four ribs done instead of seven.

Fig. 7.—Photograph of patient whose roentgenograms are shown in figure 4. Note the lack of deformity and the type of healing.

CASE 8.—W. F., a man, aged 25, had tuberculosis for two years. He was well nourished and apparently a good risk. Roentgenograms showed many small cavities throughout the upper part of the left lung with fibrosis and a much thickened pleura. The right lung was clear. Following a first stage thoracoplasty on the lower five ribs, the patient had a stormy convalescence with distention, vomiting and an acute dilatation of the stomach. The second stage was delayed unusually long, and a phrenicotomy was done in the interval. For the first four days following the second stage he did well, then the pulse became weak, irregular and rapid, respiration labored and shallow, and he died on the fifth day. The cause of death was not ascertained.

CASE 9.—S. N., a woman, aged 34, was well nourished and a good risk. She had been ill with tuberculosis for two years. Roentgenograms showed much cavitation in the upper part of the left lung and a very thick pleura. There was a fibrosed area of healed tuberculosis in the upper part of the right lung. Following

a first stage thoracoplasty in which the lower five ribs were resected, a severe streptococcic infection of the wound developed. The wound was opened widely, and irrigated with a surgical solution of clorinated soda (Dakin's solution), and a transfusion given. The patient improved, and a second transfusion was given two weeks after operation. Ten minutes after this was given, she died suddenly. The blood had been cross-agglutinated and was from the donor for her first transfusion. Autopsy failed to reveal a pulmonary embolism, and death seemed due to the transfusion as no other cause could be found.

SUMMARY

In a series of nine fatal cases, death was due to the operation in three, and in six it occurred some time afterward. Of the six patients who survived the operation and died later, two were extremely desperate risks and probably should never have been subjected to operation; two died of generalized tuberculosis and two died from extension of the disease to the good lung. Of the three whose deaths may be attributed to operation, one died of mediastinal flutter, one from transfusion and infected wound, and one of undetermined causes.

ACTINOMYCOSIS OF THE ABDOMEN*

LOUIS P. GOOD, M.D.
Fellow in Surgery, the Mayo Foundation
ROCHESTER, MINN.

Actinomycosis is a chronic disease caused by the fungus *Actinomyces bovis*. It is characterized by the formation of abscesses, sinuses, granulation tissue and brawny, leathery infiltration of the surrounding tissues. A positive diagnosis is made by demonstrating the sulphur granule or by demonstrating the actinomycotic lesion in tissue, microscopically. The microscopic picture of a typical granule may be described as a central mass of branching mycelia, radiating peripherally, and ending, usually, in clubs.

TABLE 1.—*Site of Primary Lesion in Sixty-Two Cases**

Site	Cases	Per Cent
Appendix	48	77.5
Appendix, sigmoid	2	3.2
Appendix, diverticulum	1	1.6
Tonsil, appendix	1	1.6
Pelvis, appendix	2	3.2
Gallbladder, appendix	2	3.2
Sigmoid	1	1.6
Right groin	2	3.2
Right upper quadrant	2	3.2
Abdominal wall	1	1.6

* If the original site is questionable, the one listed first is considered the more likely.

I have been unable to substantiate or contradict the two current theories of the mode of infection: (1) that the organism is carried into the tissue by some foreign material like straw and (2) that the organism is already present in the mouth and that an injury, such as a straw is likely to cause, serves to provide a portal of entry. The source of the disease in this series was evident in only a few cases. The disease was considered abdominal in type when the abdominal wall or the abdominal viscera was involved. Thirty-six per cent of the patients were farmers.

The site of the lesion (table 1) in the intestinal tract was predominantly the ileocecal region. In forty-eight cases (77.5 per cent) the lesion was apparently primary in this region, whereas in eight other cases (13 per cent) it was questionably in this region.

It would seem reasonable to assume that the fungus may be caught and harbored at a point of stasis, such as at the ileocecal region, or

* Submitted for publication, Nov. 25, 1929.

* Abridgment of thesis submitted to the Faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of Master of Science in Surgery, June, 1929.

in a diverticulum, from which it gains entry into the intestinal wall, as a result of injury to the mucosa. Frequently, there is secondary infection which results in formation of an abscess. It is probably at this stage that the patient first experiences symptoms.

In the abdominal type of actinomycosis, symptoms may be chronic or acute. In the chronic cases, there are seldom any symptoms and, in many instances, the presence of a mass in the right lower abdominal quadrant is the first indication of a lesion. Fever, malaise, pain and tenderness usually are concomitant with secondary bacterial invasion. In a high percentage of cases, this invasion occurs early in the course of the disease. The acute form, therefore, is much more frequently encountered than the chronic form. The great frequency with which the ileocecal region is involved, together with the early secondary bacterial invasion, explains the almost stereotyped group of symptoms which is encountered at the onset of the illness in the acute form.

The physician in attendance usually finds signs of acute appendicitis and advises operation. At operation, an appendical abscess frequently is found and amply drained. The appendix may or may not be removed, depending on the lesion and the condition of the patient. After the operation, the patient, in a high percentage of cases, will make an apparently normal convalescence; the wound will heal, in the case of a ruptured appendix, in from three to four weeks. Several weeks after operation, the patient begins to feel a little under par; he may have recurrence of fever, accompanied by chilly sensations, with a distinct chill at times; he feels weak and looks toxic. A region of tenderness is noted either in the incision or at some remote place such as in the flank or the right renal region. By this time, an abscess has formed. When this is drained, a sinus develops and discharges for an indefinite period. Since the nature of the disease is not recognized, every means is utilized to bring about healing and closure of this troublesome sinus. In most cases it does close, if only temporarily. In the meantime the fungus, by direct growth, progresses along the fascia, peritoneum and muscle fibers, possibly in the direction of least resistance, leaving in its wake leathery and brawny tissue, until, perhaps, some obstacle or resistance is reached and another abscess forms. Toxic symptoms recur and are relieved by drainage of the abscess and the formation of a sinus. In a similar manner, new abscesses and sinuses form while old ones may reopen, throughout the entire course of the disease. As the disease spreads by direct growth, involvement of the abdominal viscera and contiguous structures is rather extensive.

Late in the course of the disease, the blood stream may become infected and metastatic abscesses may be found in any part of the body. It is said that actinomycosis does not spread by way of the lymphatics except by direct growth. Complications (table 2) usually found are: perinephric, nephric, hepatic, subphrenic and rectal abscesses; fecal

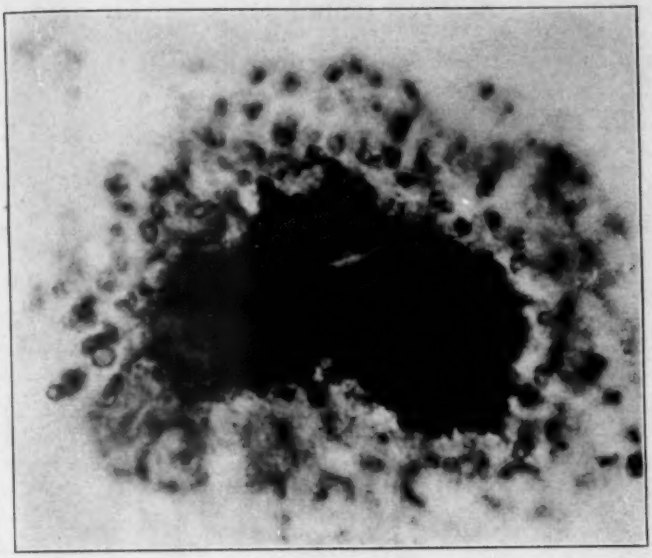


Fig. 1.—Actinomycotic node in omentun (hematoxylin and eosin; × 350).

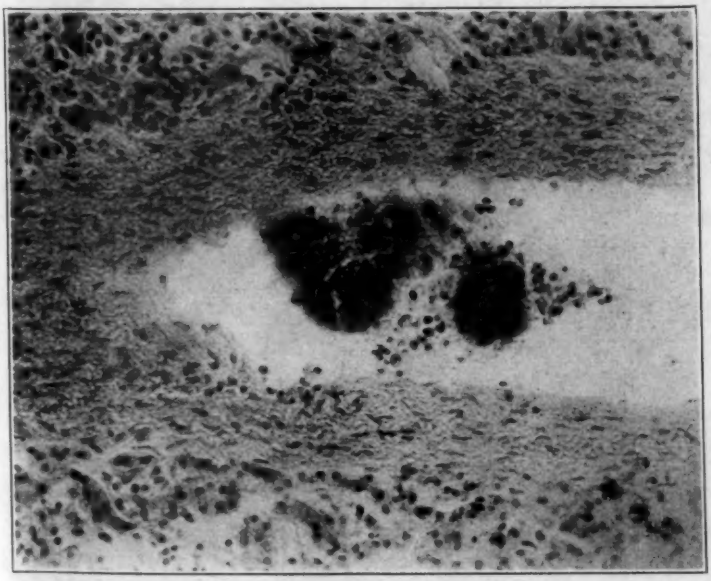


Fig. 2.—Actinomycotic abscess of the liver; thrombus of vessel is shown (hematoxylin and eosin; × 150).

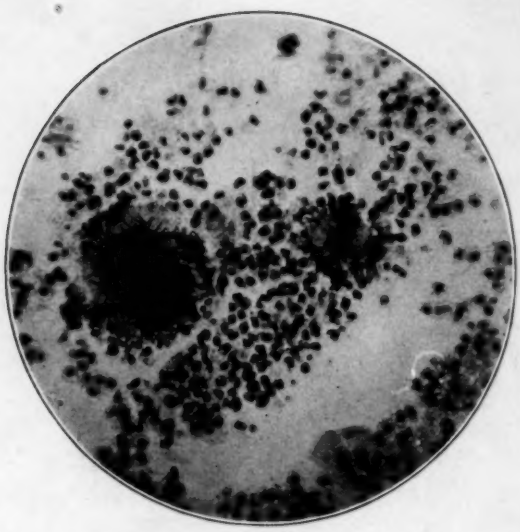


Fig. 3.—Typical actinomycotic lesion. Mycelial threads and the indistinct outline of the clubs are shown (methyl violet; × 300).

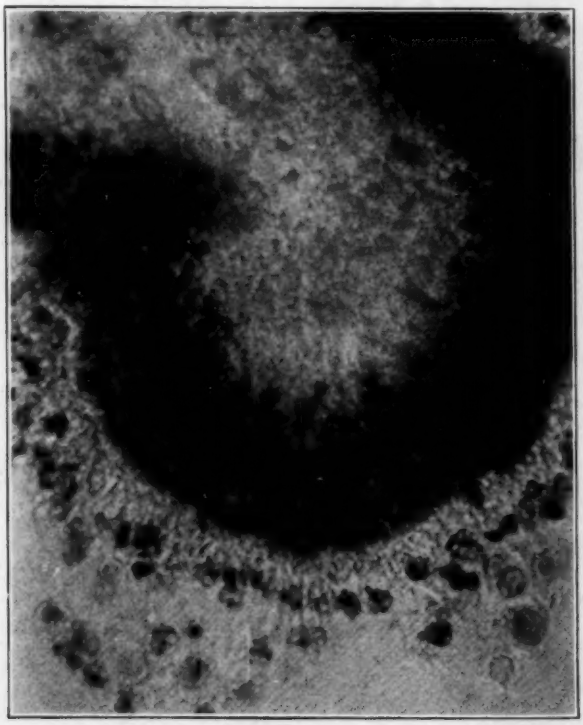


Fig. 4.—Actinomycotic lesion; clubs at the periphery of the lesion are shown (hematoxylin and eosin; × 550).

fistula; infiltration of the psoas muscle; pelvic masses, and extension to the hip, groin and abdominal wall. The bladder was involved in three cases of the series observed, giving rise to dysuria, frequency and burning, and in one case, a vesical fistula had formed. The lung is secondarily involved in 25 per cent of the cases of abdominal actinomycosis. If the lesion is primary in the ileocecal region, the lung is affected in 20 per cent of the cases; if the lesion is primary in the upper part of the abdomen, the lung is affected in 50 per cent of the cases.

In treatment, it is especially important to instruct the patient to observe every precaution to prevent the spread of the disease, not only in his own body but to other persons.

Probably the greatest hope in the treatment of patients with abdominal actinomycosis lies in its early recognition; even then it is arrested with difficulty. Treatment used at the Mayo Clinic at present may

TABLE 2.—*Structures Involved Secondarily in Sixty-Two Cases*

Structures	Cases
Lungs	15
Liver	11
Right renal	8
Pelvis	4
Abdominal wall	4
Right psoas	3
Groin	3
Bladder	3
Fecal fistula	3
Right hip	2
Rectal abscess	1
Rectal mass	1
Veins (phlebitis)	1
Scrotum	1
Iliac fossa	1
Spine	1

be surgical or medical or roentgen ray and radium may be employed. A combination of the methods is frequently used to advantage. Surgical methods are indicated for incision and drainage of abscesses, curettage of sinuses, and for the establishment of permanent drainage. In rare instances, radical operation has been effective. Medical treatment is directed to upbuilding of general health. The patient also is given massive doses of a saturated solution of potassium iodide, and the dosage is increased to the point of tolerance. Chitty has used tincture of iodine given in milk with good results. Application of roentgen ray and radium is a highly specialized form of treatment, and in the hands of competent men has given gratifying results.

Of sixty-two cases of actinomycosis of the abdomen that have been studied, twenty-nine patients have died; seven have not improved; six have improved; follow-up data could not be obtained concerning twelve, and the disease apparently has been arrested in eight.

Table 3 gives the date of onset, the duration of symptoms in terms of months both prior to registration and until death, and the treatment received, in the twenty-nine cases in which death was caused

by actinomycosis. The average duration of symptoms prior to registration was fifteen months. The average duration of symptoms from onset of the disease to death was twenty-one months. There were two

TABLE 3.—*Summary of Twenty-Nine Cases in Which Death Occurred*

Case	Date of Onset	Duration of Symptoms Prior to Registration, Months	Duration of Symptoms to Death, Months	Treatment
1	5/25	6	13	Roentgen ray, radium, potassium iodide
2	5/24	6	22	Radium, potassium iodide
3	3/16	8	10	Potassium iodide, neoarsphenamine
4	8/15	13	15	Drainage, roentgen ray, potassium iodide
5	1/16	6	?	Roentgen ray, radium, potassium iodide
6	1/17	12	12	General measures
7	1/20	7	12	Potassium iodide, roentgen ray
8	10/19	17	29	Potassium iodide, radium
9	4/19	24	26	Radium, roentgen ray, potassium iodide
10	12/24	3	7	Roentgen ray, potassium iodide
11	11/14	11	11	Potassium iodide, general
12	6/09	3	88	Roentgen ray, potassium iodide
13	6/17	3	?	Roentgen ray, radium, potassium iodide
14	11/16	11	1	General
15	3/16	1	2	Roentgen ray, potassium iodide
16	7/18	12	19	General, potassium iodide
17	3/23	6	9	Potassium iodide, copper sulphate
18	11/20	18	25	Roentgen ray, radium, potassium iodide
19	12/19	23	25	Roentgen ray, potassium iodide
20	2/26	0.5	5	Drainage of appendix, roentgen ray, potassium iodide
21	4/17	11	24	Aspiration, potassium iodide
22	5/22	11	13	Potassium iodide
23	3/22	7	10	Potassium iodide
24	11/22	11	24	Potassium iodide, roentgen ray
25	12/15	18	19	Potassium iodide
26	/06	180	211	Roentgen ray, potassium iodide
27	7/21	6	7	Potassium iodide
28	5/19	3.5	45	Potassium iodide
29	5/22	5	21	Potassium iodide
Average duration		15.3	21	

TABLE 4.—*Summary of Eight Cases Considered Arrested*

Case	Date of Onset	Duration of Symptoms Prior to Registration, Months	Total Duration of Symptoms, Months	Disease Arrested, Months	Onset to Last Follow-Up, Data, Months	Treatment
1	10/16	4	18	106	124	Sodium iodide intravenously, potassium iodide
2	6/17	4	17	96	113	Radical excision of colon and abdominal wall; roentgen ray and radium; potassium iodide
3	10/20	26	31	31	62	Roentgen ray, radium, potassium iodide
4	3/21	11	37	44	81	Roentgen ray, radium, potassium iodide
5	1/24	8	23	6	29	Roentgen ray, radium, potassium iodide
6	3/25	7	8	3	11	Roentgen ray, radium, potassium iodide
7	3/25	1	3	26	29	Incision of abdominal abscess; roentgen ray, radium, potassium iodide
8	4/25	11	14	30	44	Roentgen ray, potassium iodide

cases of long duration, cases 12 and 26, noted in table 4. If these cases are excluded, the average duration of symptoms from onset to registration would be nine months, and from onset to death fourteen and a

half months. The exact date of death could not be ascertained in three cases. These were excluded in making the computation of average duration of symptoms from onset to death. In nine of the cases (21 to 29 inclusive, table 4) the disease had extended to the lungs. If these are excluded, the average duration of symptoms from onset to registration is nine and a half months, and from onset to death, twenty and a half months.

There are eight cases in which the disease apparently has been arrested (table 4).

SUMMARY

The early symptoms of actinomycosis of the abdomen are those usually associated with acute appendicitis. Later, the disease is characterized by multiple abscesses and persistent sinuses, about which the tissue is leathery and brawny. The treatment consists in free drainage, the administration of potassium iodide, upbuilding of the general health and proper treatment by roentgen ray and radium. The course of the disease is usually chronic and the ultimate result is poor (figs. 1 to 4).

TRAUMA TO THE INTESTINES

THE IMPORTANCE OF THE LOCAL LOSS OF FLUID IN THE PRODUCTION OF LOW BLOOD PRESSURE *

ALFRED BLALOCK, M.D.
NASHVILLE, TENN.

Of the different methods that have been employed in producing "shock" in experimental animals, probably the most frequently used has been that of trauma to the intestines. Mann [1] stated, "The easiest and most certain method of producing shock is by exposure and traumatization of the abdominal viscera. This, judging from the literature, has been the method used by nearly all investigators of shock." Several investigators have gone so far as to state that this is the only method by which shock can be produced experimentally. However, during the past ten years, the method of traumatizing large areas of skeletal muscle as described by Cannon and Bayliss [2] has been used probably with equal frequency.

It has been commonly believed by many that "shock," regardless of the method of its production, is associated with an accumulation of blood in the capacious splanchnic area. Wallace, Fraser and Drummond,[3] in many operations on wounded men, did not find such to be the condition. They stated, "We would, therefore, state that in the course of many hundreds of abdominal operations undertaken upon patients in all degrees of traumatic shock we have not found any primary splanchnic congestion to exist." When the abdominal contents had been exposed to the atmosphere for a prolonged period or when an inflammatory process had become established, they found what they considered to be a secondary vascular dilatation. Many observers have commented on the congestion that occurs in the splanchnic area when the intestines are irritated. Morison and Hooker [4] found that an isolated loop of gut that was wrapped loosely in rubber tissue gained in

* Submitted for publication, March 17, 1930.

* From the Department of Surgery of Vanderbilt University.

1. Mann, F. C.: The Peripheral Origin of Surgical Shock, Bull. Johns Hopkins Hosp. **25**:205, 1914.

2. Cannon and Bayliss: Note on Muscle Injury in Relation to Shock. Report of Shock Committee of the British Medical Research Committee, March, 1919, no. 26, pp. 19-23.

3. Wallace, Cuthbert; Fraser, John; and Drummond, Hamilton: The Distribution of Blood in Traumatic Shock, Lancet **2**:727, 1917.

4. Morison, R. A., and Hooker, D. R.: The Vascular Tone and the Distribution of the Blood in Surgical Shock, Am. J. Physiol. **37**:86, 1915.

weight in the terminal stages of shock. Mann [1] stated that the factors involved in the reaction of the intestines to irritation were the same as those involved in any other local inflammatory process, and that they did not involve the central nervous system to any greater extent. The observations of the Shock Committee of the British Medical Research Committee [5] on traumatic shock supported the theory that shock is produced by histamine or a closely related substance that produces a general bodily effect.

The results of work reported in a previous communication [6] indicate that the low blood pressure which results after trauma to an extremity of the experimental animal is due to the loss of blood into the traumatized area. These experiments did not include a determination of the results that are obtained many hours after mild trauma, but only the effects that are obtained in the first few hours after rather severe trauma. No evidence for the action of a histamine-like substance was obtained. The present study was undertaken in order to try to determine whether the low blood pressure that is produced by trauma to the intestines is due to agencies that exert a general bodily effect or whether it is due simply to local alterations in the traumatized area.

METHOD

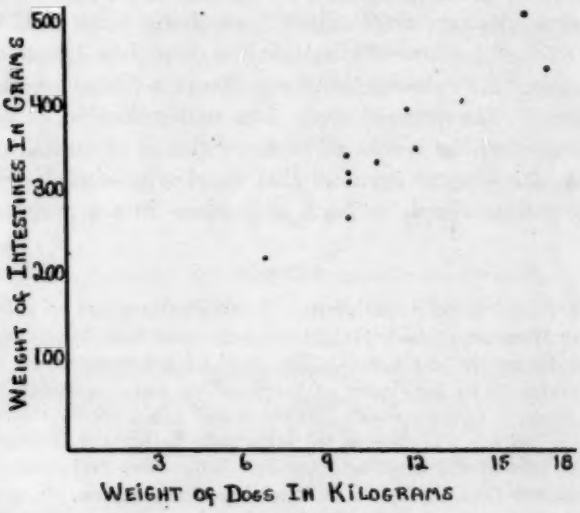
Dogs were used in all experiments. A sufficient amount of sodium barbital (0.3 Gm. per kilogram of body weight) was given to keep the animal thoroughly anesthetized during the experiment. The level of the mean blood pressure was used as the criterion of the degree of shock. This was determined by placing in the carotid artery a cannula which was connected to a mercury manometer. The weight of the animal was determined accurately before the traumatization was begun. That part of the small intestine that is between Treitz ligament and the appendix was delivered through a midline abdominal incision. Gauze sponges of known weight were placed under the intestines in order to collect the fluid that escaped from the visceral peritoneum. The intestines were traumatized in ten of the twelve experiments by pinching them with the fingers. In the remaining two experiments, the trauma consisted of pulling repeatedly on the mesentery of the exposed intestines. The trauma was intermittent, the average total duration of the trauma in the different experiments being three hours and twenty-five minutes. When the gauze that was placed beneath the intestines became saturated with fluid, it was placed in a container that prevented evaporation and it was replaced by dry sponges. At the completion of the experiment, the sponges were again weighed, and the difference between the weights of the sponges at the beginning and at the termination of the traumatization was determined. After the decline in blood pressure that was desired had been produced, the dog was again weighed, and the loss in weight of the entire animal was determined. The part of the intestinal tract that had been traumatized was removed, after clamps had been

5. Reports of Shock Committee of the British Medical Research Committee, nos. 25, 26 and 27, 1919. Published by His Majesty's Stationery Office, London.

6. Blalock, Alfred: Experimental Shock: The Cause of the Low Blood Pressure Produced by Muscle Injury, Arch. Surg. **20**:959 (June) 1930.

placed across its pedicle. The weight of this was determined. The animals were not fed on the morning of the day of the experiments, and there were practically no feces in the small intestine.

In order to obtain an approximate idea of the weight of a similar portion of the intestinal tract when no trauma had been instituted, the weight of the intestines between Treitz' ligament and the appendix was determined in eleven dogs. These animals were anesthetized by barbital or ether. This portion of the intestines was removed immediately after opening the peritoneal cavity, and the weight was determined. From these figures and those for the weights of the dogs, the average weight of the intestinal tract per kilogram of body weight was determined. This average figure was multiplied by the weight of the dogs that were traumatized in order to gain an approximate idea as to what the intestines weighed before the trauma was instituted. The average weight of the intestines per kilogram of body



The weight of the excised intestine in eleven dogs in which no trauma was instituted. The weight of the intestines in grams is plotted against the weight of the dogs in kilograms.

weight in the control experiments was 30.7 Gm. The weights in individual experiments are shown in the chart.

The figure that was chosen as giving roughly the amount of available fluid that was lost from the circulating blood volume was obtained by adding the difference in the weight of the dog at the beginning and the end of the trauma to the difference between the determined weight of the traumatized intestine and the estimated weight of the nontraumatized intestine of a dog of the same weight. The sum of these figures divided by the weight of the dog gave the percentage of the body weight that was lost.

The hemoglobin of blood obtained from the femoral vein was determined before the trauma was begun and at its termination. After the intestines had been removed and weighed, they were placed in salt solution. They were incised in many places, and an attempt was made to wash out all of the blood that they contained. The amount of whole blood that was present in the washings was

obtained by multiplying the percentage of hemoglobin of the fluid by the total amount of the fluid and dividing by the percentage of hemoglobin of the whole blood. The amount of blood that had been collected by the sponges was determined in the same way.

RESULTS

The interval that elapsed between the initiation of the trauma and the reduction of the blood pressure to the desired level varied in the different experiments from three hours and ten minutes to eight hours. The mean blood pressure at the time that the trauma was discontinued varied from 26 to 68 mm. of mercury. Trauma to the intestines resulted in a concentration of the blood as revealed by the determinations of hemoglobin. The average increase in the percentage of hemoglobin in all experiments was 38 per cent.

The sponges that were placed under the intestines in order to collect the secretions gained in weight from 125 to 520 Gm. in the different experiments. This difference in weight of the sponges in most instances represented about one-half of the difference between the weight of the animal at the beginning and that at the termination of the trauma. This discrepancy is explained in part at least by the fact that part of the fluid evaporated from the surface of the intestines and from the sponges. The difference in the weight of the dog at the beginning and the end of the experiment varied in the different experiments from 195 to 690 Gm. This indicates that fluid lost from the body in the different experiments varied approximately from 195 to 690 cc. The average weight of the dogs was 12.18 Kg., and the average loss of fluid from each animal was 433 cc. A small part of this can be accounted for by that lost in the expired air. This is relatively unimportant, as it was found in control experiments that dogs weighing 12 Kg. lose only about 60 Gm. when observed for a period corresponding to the average duration of the experiments in which the intestines were traumatized. Precautions were observed in all experiments to guard against false values obtained by loss of urine or feces.

The weight of the traumatied intestine was greater than that of the estimated weight of the intestine before trauma by amounts varying from 11 to 233 Gm. In view of the fact that the weight of the normal intestine had to be computed from figures obtained on dogs other than those that were traumatied, the variations are not surprising. The average increase in weight of the intestines as computed was 103 Gm.

It was believed that the difference between the weight of the animal at the beginning and that at the termination of the trauma plus the difference between the weight of the traumatized intestine and its estimated normal weight would give an approximate idea as to the combined losses of fluid from the blood stream and the body tissues. This loss in the different experiments varied from 206 to 878 Gm., and the

Experiment	Control Blood Pressure, Mm. Hg	Blood Pressure Before Amputation, Mm. Hg	Time Between Initial Trauma and Amputation	Control Hemoglobin, per Cent	Hemoglobin Before Amputation, per Cent	Initial Weight of Sponges, Gm.	Weight of Sponges Before Amputation, Gm.	Difference in Weight of Sponges, Gm.	Estimated Initial Weight of Intestines, Gm.	Weight of Intestines at Amputation, Gm.	Difference in Weight of Intestines, Gm.	Control Weight of Dog, Kg.	Weight of Dog Before Amputation, Kg.	Difference in Weight of Dog, Gm.	Loss of Weight in Dog Plus Gain in Weight of Intestine, Gm.	Percentage of Body Weight	Blood From Intestine, Cc.	Blood From Sponges, Cc.
1	103	54	5 hr. 30 min.	67	100	200	340	140	392	500	108	12.81	12.49	320	428	3.34	57	25
2	100	34	3 hr. 15 min.	80	130	180	340	160	472	575	103	15.30	15.1	200	303	2.55	99	43
3	104	63	3 hr. 10 min.	61	105	270	500	230	377	610	233	12.295	11.985	310	543	4.41	43	55
4	91	58	6 hr. 50 min.	?	90	200	455	255	271	405	134	8.83	8.415	415	549	6.22	70	93
5	138	47	3 hr. 40 min.	88	103	220	365	145	275	340	65	8.97	8.70	270	335	3.73	39	88
6	144	65	3 hr. 30 min.	96	108	225	350	125	334	345	11	10.895	10.70	195	206	1.89	47	41
7	120	26	5 hr. 0 min.	83	88	265	785	520	441	530	89	14.37	13.68	690	779	5.42	88	500
8	98	68	4 hr. 20 min.	55	100	275	650	375	485	575	90	15.805	15.26	545	635	4.02	38	26
9	113	29	6 hr. 25 min.	60	67	380	745	365	437	655	218	14.24	13.58	660	878	6.16	168	144
10	116	63	8 hr. 0 min.	60	82	270	450	180	292	320	28	9.52	9.08	440	468	4.92	...	...
11*	90	38	4 hr. 10 min.	?	76	235	380	145	282	480	198	9.18	8.85	330	528	5.75	45	60
12*	112	34	6 hr. 10 min.	60	90	330	725	395	431	450	19	14.045	13.31	735	754	5.39	37	39

* Trauma consisted entirely of pulling on mesentery. In all other experiments, it consisted of pinching intestines.

average loss was 541 Gm. The loss in fluid expressed in percentages of body weight varied in the different experiments from 1.89 to 6.22 per cent, and the average loss was 4.48 per cent. In ten of the twelve experiments, the calculated loss represented more than 3 per cent of the body weight, and in eight of the twelve experiments, it represented more than 4 per cent. Determinations of the amount of whole blood that was present in the sponges showed that the red cells in most experiments constituted a relatively small proportion of the total loss. It was obviously impossible to recover all of the blood from the intestines, and hence no conclusion could be reached as to the relative parts played by plasma and red cells in the increase in weight.

The results that were obtained in all of the experiments on intestinal trauma are given in the accompanying table.

As has been stated previously, the intestine that was to be traumatized was pulled outside the peritoneal cavity through the incision in the anterior abdominal wall. It is interesting that although there was a copious loss of fluid from the intestine that was traumatized, there was no demonstrable loss of fluid from the organs that were left in the peritoneal cavity. Neither did these organs appear macroscopically to be engorged with blood. The walls of the gallbladder did not become edematous, and they retained their bluish color. In all experiments, the spleen decreased greatly in size during the traumatization. The weights were not determined, but the decrease was estimated at 50 per cent. In some instances, blood was found in the lumen of the large intestine at the completion of the experiment. The amount of this was not determined, and it was neglected in determining the fluids that were lost from the circulation.

In several additional experiments, the effects of passing a stream of compressed air over the exposed intestines were determined. The loss in weight from evaporation was rapid. The body temperature of the animal fell quickly, and usually death resulted before as much fluid had been lost as was found in the experiments in which the intestines were traumatized.

COMMENT

Prolonged trauma to the intestinal tract resulted in a decline in the blood pressure. Similar results were obtained regardless of whether the trauma consisted of pinching the intestines or of pulling on the mesentery. The decline in blood pressure was almost certainly due to a diminution in the circulatory blood volume, as the preponderance of evidence at the present time indicates that there is a vasoconstriction at least during the early stages of the shock. There are several possibilities as to the manner in which the blood volume was reduced: (1) hemorrhage outside the body or into the tissues; (2) loss of plasma

from the blood stream by filtration, with a concentration of the blood, and (3) stasis of blood in the traumatized area. External hemorrhage was measured with moderate accuracy by collecting all of the blood that was absorbed by the sponges. This constituted a small part of the total loss of fluid. The blood pressure was determined at infrequent intervals, and small cannulas were used, so that the amount of blood that was lost in the "washings" amounted to only a few cubic centimeters. There was a great loss of fluid from the blood stream by filtration, as determined by the increase in the weight of the sponges and by the difference in the weight of the animal at the beginning and the end of the experiment. The marked increase in the concentration of the blood in most experiments indicated a tremendous loss of plasma. Determinations of the hemoglobin indicated the minimum loss of blood through filtration of the plasma, as any local retention of the red cells would decrease the percentage of hemoglobin of the blood from the femoral vein. In several of the experiments, the large losses of fluid and the relatively small increase in the hemoglobin indicated that stasis was playing a detectable rôle in the diminution in the circulating blood volume. That there was stasis in the traumatized area is indicated by its weight being greater than that of the estimated normal. This observation is confirmatory of those of Morison and Hooker,[4] who showed that an exposed and isolated loop of the intestine increased in weight in the terminal stages of shock.

Gasser, Erlanger and Meek [7] determined the protein content of the plasma of the blood after clamping the aorta. They found that the protein content of the plasma underwent no marked change during the process of the concentration of the blood and believed that the plasma was lost mainly as a whole. In uncomplicated hemorrhage in small amounts, there is a passage of fluid from the tissue spaces into the blood stream. Starling [8] explained this as being due to reduction of the blood pressure in the capillaries, so that the filtration pressure within them no longer effects the greater osmotic pressure of the plasma as compared with the lymph, and water passes into the blood stream. There is doubt whether or not water passes into the blood stream when a low blood pressure is produced by trauma. Cannon [9] stated, "It is one of the unexplained features of shock that with a low venous and arterial pressures this process does not occur. Instead, the plasma as a whole makes its escape through the vessel walls." The fluid that escaped from the intestines in the present study was not analyzed. This

7. Gasser, H. S.; Erlanger, Joseph; and Meek, W. J.: Studies in Secondary Traumatic Shock: IV. The Blood Volume Changes and the Effect of Gum Acacia on Their Development, Am. J. Physiol. **50**:31, 1919.

8. Starling: Fluids of the Body, Chicago, W. T. Keener & Company, 1909.

9. Cannon: Traumatic Shock, New York, D. Appleton & Company, 1923.

will form the subject of future study. In view of previous work, it seems most likely that the plasma made its escape as a whole. Possibly at the same time that plasma was being lost from the blood stream in the injured area, fluid was passing from the tissue spaces into the blood stream in the uninjured parts of the body. This exchange cannot continue indefinitely, since the available fluids in the body tissues are soon consumed. If the fluids that were lost from the surface of the visceral peritoneum in the present experiments consisted in the main of plasma, it is evident that there must have been a tremendous reduction in the circulating volume of blood. In a large series of dogs, Gasser, Erlanger and Meek [7] found the average blood volume to be 9.7 per cent of the body weight. They estimated that the plasma constitutes 60 per cent of the volume of the blood. The calculated average loss in weight in the experiments with intestinal trauma was 4.48 per cent of the body weight. If it is assumed for the moment that all of the fluid that was lost was plasma and that it was not replaced by fluid from the tissues, it is to be seen that 77 per cent of the total plasma was lost. This is probably impossible, for in such a high concentration the red blood cells most likely would not circulate. It is likely, however, that the loss of plasma was sufficient to cause so much concentration of the blood that the internal friction of the corpuscles against the walls of the blood vessels was greatly increased. Concerning the effects of concentration of the blood, Cohnheim [10] stated, "The concentration of the blood due to water inanition is evidently too slight to affect the heart and vessels to any considerable extent. As regards the withdrawal of water by artificially increasing the diffusion into the abdominal cavity, we know only that it may prove fatal to rabbits, but are ignorant of the effects of this condition when prolonged." He believed the effects of the concentration to be first and chiefly an enormous increase of the frictional resistance in the capillaries. Trevan [11] found that the internal friction rises rapidly when the concentration of the corpuscles is increasing. The stagnation of the blood that is caused by the concentration is accentuated by the decrease in arterial pressure that results from the loss of fluid from the blood stream.

It has been shown by Blalock and Harrison [12] that normal dogs usually reach a state of severe shock after blood amounting to from 3 to 4 per cent of the body weight has been removed. A comparison of the deleterious effects of the removal of equal amounts of whole

10. Cohnheim: Lectures in Pathology, London, The New Sydenham Society, vol. 1, p. 465, 1889.

11. Trevan: Biochem. J. **12**:60, 1918.

12. Blalock, A., and Harrison, T. R.: The Regulation of the Circulation: V. The Effect of Anemia and Hemorrhage on the Cardiac Output of Dogs, Am. J. Physiol. **80**:157, 1927.

blood and of plasma has not been satisfactorily determined. Kallius[13] stated that the serum and not the red blood cells plays the more important rôle in the replacement of an acute loss of blood. Abel, Rowntree and Turner[14] found that it was possible to withdraw by repeated bleedings in a single day a volume of blood more than twice that contained in the body, with no apparent injury to the animal, if only the corpuscles suspended in Locke's solution were returned after each bleeding. In the present experiments on intestinal trauma, there was no replacement of the fluid that was lost. It seems likely that the loss of plasma from the blood stream amounting to 4 per cent of the body weight would produce an effect approximately as grave as the loss of the same amount of whole blood. It has been stated that the calculated loss of fluid in eight of the twelve experiments amounted to more than 4 per cent of the body weight. In these experiments, it is believed that the alterations in the traumatized area alone were sufficient to account for the reduction in the blood pressure. The loss of fluid from and the accumulation of blood in the traumatized area result in a reduction in the blood pressure. Operating to sustain this decrease in pressure are the increase in the viscosity of the blood, the elevation of the internal friction of the corpuscles against the walls of the vessels, the decrease in the amount of blood that returns to the heart and the decrease in the cardiac output. Erlanger, Gesell and Gasser[15] believed that the loss of fluid into and through the tissues of the bowel and the sequestration of blood in the intestinal capillaries and venules suggested a mechanism through which a reduction in blood volume might occur. Excision of the stomach, spleen and intestines led them to believe that this was not the entire mechanism. They stated, "But if this is the mechanism, the fact that after excision of the stomach, intestines, and spleen the arterial pressure falls almost exactly in the same way as after exposure of and manipulation of the intestines, and the fact that the changes in peripheral resistance are also alike, necessitate assuming that blood is thus removed from circulation, not alone in the parts of the body directly traumatized, but elsewhere also." In the three experiments that they performed, the animals were anesthetized by ether, and death followed the removal of the stomach, intestines and spleen at periods of time ranging from four and one half to seven hours. These experiments do not prove that the decline in blood pressure following

13. Kallius, H. W.: Experimentell Untersuchungen über die Wirkung des Serums bei der vitalen Bluttransfusion, Deutsche Ztschr. f. Chir. **212**:289, 1928.

14. Abel; Rowntree; and Turner: Plasma Removal with Return of Corpuscles (Plasmaphaeresis), J. Pharmacol. & Exper. Therap. **5**:625, 1913-1914.

15. Erlanger, Joseph; Gesell, Robert; and Gasser, H. S.: Studies in Secondary Traumatic Shock: I. The Circulation in Shock after Abdominal Injuries, Am. J. Physiol. **49**:90, 1919.

traumatization of the intestines is due to factors other than the loss of fluid from and the accumulation of blood in the injured area. The removal of the stomach, intestines and spleen is necessarily associated with the removal of a considerable part of the total volume of blood. Although this is not followed in their experiments by an immediate decline in blood pressure, it almost certainly renders the animal more liable to a drop in pressure during prolonged ether anesthesia. They do not state whether or not the peritoneum was closed. If the peritoneum was left exposed to air, undoubtedly there was a considerable loss of fluid from it. Even if the incision was closed, there was probably a moderate amount of loss of fluid from the injured peritoneum. No mention is made as to the presence or absence of fluid in the peritoneal cavity at the time of death.

In two of the twelve experiments, the calculated loss of fluid from the blood stream amounted to less than 3 per cent of the body weight. In one of these, the mean blood pressure was still rather high (65 mm. of mercury) at the time the experiment was terminated. The cause for the decline in pressure was undertermined. It seems unlikely that it was due entirely to the loss of fluid from the traumatized area. It is possible that it was due in these two exceptional experiments to the barbital, for it has been found in a large series of experiments that the giving of barbital without producing trauma is occasionally associated with a slow decline in the blood pressure.

The amount of fluid loss that can be tolerated without producing a marked decline in the blood pressure is probably influenced by several factors. In the experiments in which a stream of cool air was blown over the intestines, the temperature of the animal fell rapidly. Death was caused more quickly and with less loss of fluid than in the experiments in which the intestines were traumatized. Barbital seems to render the heat regulating mechanism of the body unstable, and possibly this was partially responsible for the difference.

Gasser, Erlanger and Meek [7] studied shock produced by the injection of large doses of epinephrine hydrochloride, by occlusion of the aorta or vena cava and by exposure and manipulation of the intestines. As regards the effects of these procedures on the spleen, they stated, "The spleen is usually swollen and has dark raised areas which consist of hemorrhages into the pulp. This swelling and hemorrhage in some cases produce a spleen many times the normal size." These experiments were performed on dogs. Moore [16] produced shock in various ways in cats and determined the size of the spleen. Ten experiments were performed. In six of these, shock was produced by crushing of

16. Moore, R. M.: The Volume of the Spleen in Traumatic Shock, Am. J. Physiol. **89**:508, 1929.

the thigh muscles and the testicles; in three, the peritoneum was opened, and the cavity was inflated with air for two hours at a pressure equal to 40 cc. of water, and in the remaining experiment, the animal was cooled. After a low blood pressure had been produced by each of these procedures, the size of the spleen was found to be reduced. In regard to the condition of the splanchnic blood vessels in shock, he stated, "The close relationship which exists between the state of contraction of the spleen and that of the splanchnic vessels in general seems to justify the conclusion that in the experiments reported the splanchnic vasoconstrictor mechanism was operating powerfully and efficiently at a time when the blood pressure was below the critical level." Moore made the reservation that exposure or manipulation of the abdominal viscera leads to local congestion. This, he believed, is a local response to the trauma and is not due to a paralysis of the vasoconstrictor center. The size of the spleen was observed at the beginning and at the termination of the present experiments on dogs. In all instances there was a tremendous decrease as a result of the trauma. Apparently, the vessels of the traumatized intestine were dilated and those of the spleen were constricted. A vasoconstriction probably existed in that part of the intestinal tract that was not traumatized, as no engorgement with blood was apparent and there was no detectable weeping from the peritoneal surfaces.

It was found by Bradburn and Blalock[17] that histamine, when injected in sufficient amounts to produce a sustained decrease in the blood pressure, caused edema of the wall of the gallbladder. In the present experiments, the gallbladder appeared entirely normal, indicating that histamine was not liberated in sufficient amounts from the traumatized area to account for the decline in pressure.

SUMMARY

The effects of trauma to the intestines have been studied in twelve dogs. The loss of fluid from the body and into the traumatized area of the intestines has been determined. This loss in eight of the twelve experiments amounted to more than 4 per cent of the body weight. The average loss in all experiments was 4.48 per cent of the body weight. Red blood cells constituted only a small part of the fluid that escaped from the peritoneum covering the intestines. Reasons are given for the belief that the loss of fluids from and into the traumatized area was the chief if not the sole cause for the reduction in the blood pressure.

17. Bradburn, H. B., and Blalock, A.: Unpublished Observations on the Effects of Histamine on the Gall Bladder, 1929.

FORTY-THIRD REPORT OF PROGRESS IN ORTHOPEDIC SURGERY *

PHILIP D. WILSON, M.D.

LLOYD T. BROWN, M.D.

M. N. SMITH-PETERSEN, M.D.

JOHN G. KUHNS, M.D.

AND

EDWIN F. CAVE, M.D.

BOSTON

RALPH K. GHORMLEY, M.D.

ROCHESTER, MINN.

MURRAY S. DANFORTH, M.D.

PROVIDENCE, R. I.

GEORGE PERKINS

LONDON, ENGLAND

ARTHUR VAN DESSEL, M.D.

LOUVAIN, BELGIUM

AND

C. HERMANN BUCHOLZ, M.D.

HALLE, GERMANY

CONGENITAL DEFORMITIES

Congenital Clubfoot.—Brockman,[1] in the Robert Jones prize essay, advocated the correction of rigid and relapsed types of clubfoot by an open operation, based on the conception that the deformity was due primarily to a congenital atresia of the socket for the head of the astragalus. The operation aimed at making this socket sufficiently large for the head of the astragalus to be replaced in its normal position, and in addition the muscles, which controlled the variations in the capacity of the socket, were lengthened. Under an Esmarch bandage an incision was made on the outer side of the foot over the os calcis. Through this the plantar muscles and fascia were detached as far backward and inward as possible. A second incision was made on the inner side of the foot, and the remaining attachments of these muscles were completely freed from their origin. The origins of the abductor hallucis from the sheaths of the tendons and the vessels passing into the sole of the foot were dis-

* This Report of Progress is based on a review of 206 articles selected from 469 titles dealing with orthopedic surgery appearing in the medical literature between May 1, 1930, and Sept. 15, 1930. Only those papers that seem to represent progress have been selected for review.

1. Brockman, E. P.: Congenital Club-Foot (Talipes Equinovarus), Bristol, John Wright & Sons, 1930.

sected off. The tendon of the tibialis posticus was then identified. If this prevents the calcaneoscaphoid joint from being identified, it should be detached from the tubercle of the scaphoid and, if necessary, its accessory attachments going to the other bones of the tarsus divided. The next step was to dissect the structures off the inferior surface of the tarsal bones until the whole of the inferior and internal surfaces of the scaphoid and the inner aspect of the sustentaculum tali were exposed. All the ligaments on the inner and inferior aspects of the calcaneoscaphoid joint were then divided. It was then possible to restore the forefoot to its normal position in relation to the astragalus. The foot was not put up in full correction, owing to possible interference with the circulation. At the end of fourteen days, under another anesthetic, full correction was obtained by forcible manipulation and the tendo achillis elongated, if a true equinus was present. After from six to eight weeks in plaster ordinary boots were supplied, a clubfoot shoe ordered to be worn at night, and reeducation of the evertor muscles was commenced. The author did not favor operating on patients under the age of 3, because of the large amount of fat and the smallness of the structures in the sole.

[Ed Note.—We do not feel that the whole deformity of clubfoot can be so easily described. Any one who has studied Hoke's collection of astragali must have been impressed by the extreme and varied types of deformities they presented, particularly in respect to the head and neck. These must be taken into account in the correction of clubfoot deformity. Except that the author recommends a second incision along the outer border of the os calcis, we see little fundamental difference between this operation and that of Ober.[2] We prefer gentle manipulative methods of correction under the age of 3 or 4 years.]

Congenital Dislocation of the Hip.—Jaeger [3] made a plea for early recognition of congenital dislocation of the hip and described his method of treating the condition when discovered in infancy. He employed a hip splint of rustless steel which was fastened to the pelvis and chest by circular bands and to the foot and ankle by a leather anklet and foot piece. Lateral compression of the head into the acetabulum was obtained by an adjustable pad fastened to the splint and placed directly over the trochanter. By gradually abducting the thigh the head of the femur was gently pressed into the acetabulum. The splint was worn continuously for from four to eight months.

Beck [4] reported the late results of 107 cases of old congenital dislocation of the hip in Bier's surgical clinic in Berlin. In all cases reduc-

2. Ober, F. B.: An Operation for Congenital Equinovarus Deformity: Preliminary Report, J. A. M. A. **65**:621 (Aug. 14) 1915.

3. Jaeger, C. H.: Surg. Gynec. Obst. **50**:757 (April) 1930.

4. Beck, Heinz: Zentralbl. f. Chir. **56**:2582, 1930.

tion was done at or after the sixth year. The method was the typical reduction over the upper rim of the acetabulum after previous traction with wire on the lower end of the femur in order to bring the head down to the height of the acetabulum. The fixation in plaster cast was carried out for from three to six months, but not longer because of the danger of permanent fixation. After the ninth year the reduction did not succeed, except by several forced manipulations under ether. On the whole, the results had not been very good; even when the primary result was satisfactory, the end-result was not completely successful on account of the insufficient formation of the acetabular shelf. In ten cases a fracture resulted, in eight cases a solid ankylosis and in eight cases redislocation. At the age of from 12 to 15 years typical symptoms appeared in many cases of successful reduction caused by the secondary deformations of the head.

Syndactylism.—Davis and German [5] studied the cases of fifty patients with syndactylism treated at the Johns Hopkins Hospital. Eighteen per cent of the patients gave a family history of syndactylism. They were treated by skin flaps, whole thickness skin grafts and occasional plastic operations on the bones of the fingers. The authors advised against extensive plastic operations at an early age; they said that only that which was necessary to prevent disturbance in growth and increasing deformity in infancy, should be done and that the operative procedures should be completed at the age of 6 or 7.

Bifid Os Calcis.—Sever [6] reported three cases of bifid os calcis in infants. He was unable to find any reference to this in the literature. The condition was bilateral and might be mistaken for fracture. The defect usually disappeared at or before the age of 3 years.

Experiments on Embryo.—Debrunner [7] did experiments on the embryos of dogs and rabbits during intra-uterine life. These experiments showed that fetal tissue had very slight resistance against mechanical insults. Even a loose constriction of a leg with a thin silk strand led to a deformity resembling amniotic amputation. On the other hand, embryonic tissue had a marked power of regeneration, as an osteotomy of a long bone healed within four days. Parts of the extremities amputated by constriction were never found after birth, showing the marked resorptive power of the intra-uterine fluid.

METABOLIC DISTURBANCES OF BONE

Osteitis Fibrosa.—In most cases Eliason and North [8] regarded trauma as the cause of osteitis fibrosa. They were inclined to belittle

5. Davis, J. S., and German, W. J.: Syndactylism (Coherence of Fingers or Toes), Arch. Surg. **21**:32 (July) 1930.
6. Sever, J. W.: Surg. Gynec. Obst. **50**:1012 (June) 1930.
7. Debrunner, H.: Arch. f. Orthop. **28**:25, 1930.
8. Eliason, E. L., and North, J. P.: Ann. Surg. **91**:833 (June) 1930.

the infectious theory advocated by Phemister. The common locations for the lesion were pointed out as the greater tuberosity of the humerus, lower end of the radius, one or the other end of the tibia and the femur near the greater trochanter. These they stated were regions frequently exposed to trauma. For treatment they favored a combination of the roentgen rays and surgical intervention, realizing that prolonged roentgen treatment might damage the adjacent growing epiphysis, and that recurrences might follow operative treatment alone. It was essential to observe such patients over a period of several years after any form of treatment.

Osteomalacia.—Gargill and his associates [9] reported metabolic studies in a case of osteomalacia occurring in an American woman, aged 38. The patient was first placed on a diet low in calcium and vitamins, on which a normal person gave a negative calcium balance of 6.7 mg. On this diet the patient gave a negative calcium balance of 45 mg. She was given 50 cc. of a physiologically tested brand of cod liver oil and 200 Gm. of powdered skimmed milk, but no improvement was noted. Oscodol tablets (concentrated cod liver oil) were then added, and slight improvement occurred. The addition of treatment with ultraviolet light still further improved the condition. With the addition of large amounts of calcium in the form of calcium lactate, phosphorus in the form of disodium acid phosphate and a diet rich in vitamins, improvement was observed. The pain over the ribs and back, which had been the chief symptom, wholly disappeared, and progressive recalcification was observed in the roentgenograms. The authors reviewed the literature. In this case they felt that they were dealing with a form of adult rickets without any evidence of an endocrine disturbance.

Compere [10] noted the frequent association of osteomalacia and allied disorders with hyperparathyroidism. A case of osteomalacia was reported in which marked improvement occurred after removal of a parathyroid tumor. Eleven additional cases were reported from the literature. In eight of these there were tumors of one of the parathyroid glands demonstrated at operation. In five of these there was symptomatic improvement and increased deposit of calcium after removal of the tumors.

Renal Rickets.—Schoenthal and Burpee [11] reported in detail a case of renal rickets in a child of 8 years, and gave the complete metabolic studies carried out over a period of five years. According to the authors,

9. Gargill, S. L.; Gilligan, D. R., and Blumgart, H. L.: Metabolism and Treatment of Osteomalacia: Its Relation to Rickets, Arch. Int. Med. **45**:879 (June) 1930.

10. Compere, E. L.: Surg. Gynec. Obst. **50**:783, 1930.

11. Schoenthal, L., and Burpee, C.: Renal Rickets, Am. J. Dis. Child. **39**:517 (March) 1930.

two types of renal rickets might be distinguished: (1) that due to congenital malformation of the kidney or urinary tract, and (2) another type due to chronic renal insufficiency, chronic interstitial nephritis becoming manifest in later childhood. The renal insufficiency led to an acidosis which was supposed to cause mobilization of calcium from the bone resulting in decalcification.

Zanoli [12] reported two observations of his own, and collected from the literature thirty-two more cases of what he was pleased to call renal pseudorickets. The syndrome consisted of a renal lesion, skeletal deformities, especially genu valgum, and retardation of development of the body. In studying the cases, he found that in the majority the retardation of development began either at birth or in early infancy. The appearance of skeletal deformities was variable. Rarely it occurred in the first or second year of life, more often at 4 or 5 years, sometimes at 7 or 8 years and not infrequently at from 12 to 15 years. It occurred more commonly in boys than in girls. Skeletal deformities constituted one of the cardinal symptoms, being present in 67 per cent, and the most common deformity was genu valgum, often bilateral. In the great majority of cases, the renal condition was chronic interstitial nephritis. The retardation of development in stature was such that the child was from two to nine years behind other children of the same age, with an average of three or four years. Correction of deformities should be undertaken with care, as the children are poor operative risks.

[ED. NOTE.—The term renal pseudorachitism seems to us a more suitable name than renal rickets or the term recently suggested in an editorial of the *Journal of the American Medical Association* [13] "renal infantilism." Fortunately, the condition hardly seems common enough in this country to cause much concern over the nomenclature.]

GROWTH DISTURBANCES OF BONE

Effect of Roentgen Radiation.—Desjardins [14] reported a case of small round cell sarcoma of the humerus in a child in which roentgen treatment was followed by an inhibition in the growth of the right humerus and feeble development of the muscles about the right shoulder. The patient was followed up for a period of four and a half years. A continued repair of the humerus was noted, but the muscle and bone remained much smaller than the corresponding parts of the opposite side. The author reviewed the literature on retardation of growth by the roentgen rays.

12. Zanoli, R.: Chir. d. org. di movimento **14**:539 (March) 1930

13. Renal Rickets—An Undesirable Designation? Editorial, J. A. M. A. **95**:116 (July 12) 1930.

14. Desjardins, A. U.: Radiology **14**:296, 1930.

ANATOMIC STUDIES—BACK AND FOOT STRAIN

Pain in the Lower Part of the Back.—Brackett [15] divided derangements of the lower part of the back into cases with normal anatomic structure and those with anatomic malformations. In cases with normal anatomic structure, rest and fixation during convalescence were all that was required in the way of treatment. In cases with structural abnormality, the physician had to determine to what extent the abnormality interfered with the functional integrity of the lumbosacral and sacro-iliac joint, and the treatment had to be modified accordingly. The author felt that sacralization of the fifth lumbar vertebra played very little part in derangements of the lower part of the back. A horizontally placed sacrum and irregular lumbosacral articulations were potential factors in back strain, particularly because of the strain they produced on the sacro-iliac joints.

Riches [16] made a study of the end-results in 75 of 113 patients with pain in the lower part of the back, who had been treated by mobilization of the back under general anesthesia. He found the following results:

	Number	Number Improved	Per Cent
Chronic back strain	30	26	87
Sacro-iliac strain	25	23	92
Lumbosacral strain	3	1	33
Spinal arthritis	13	6	46
Neurotic spine	4	0	0

No patient was classified as improved unless he had returned to work and did not suffer more than occasional pain in the back after overexertion. After-treatment, with exercises with the intention of maintaining the mobility produced while under anesthesia, was essential. The manipulation sometimes needed repeating twice or even three times. Cases with a traumatic history gave better results (97 per cent improved) than those with an insidious, nontraumatic onset. Sixty-eight per cent of the cases classified as strain (chronic pain in the back, sacro-iliac or lumbosacral) were traumatic in origin. Roentgenograms in the cases of strain were negative, except for anatomic anomalies which appeared to be of no consequence. Stereoscopic roentgenograms in cases of spinal arthritis showed lipping of the bodies and of the articular processes sometimes. In cases designated "chronic back strain" the lesion was over a wide area, being usually a strain of the erector spinae muscles or of ligaments underlying the muscles, diagnosed clinically by tenderness over the erector spinae, spasm of the muscles and limitation of lateral flexion of the spine toward the opposite side. In cases designated "sacro-iliac strain" the history was a sudden unguarded movement giving rise to sharp pain referred to one or the other sacro-iliac joint.

15. Brackett, E. G.: J. Bone & Joint Surg. **12**:325, 1930.
16. Riches, E. W.: Lancet **1**:957 (May 3) 1930.

This was followed by a sense of insecurity. On examination there was tenderness localized to the posterior superior spine or just below. Straight knee hip flexion caused pain to be felt in the sacro-iliac joint. In cases designated "lumbosacral strain," the tenderness was localized accurately to the lumbosacral junction in the midline or just to either side of it. Flexion of the spine was free and painless, but hyperextension was painful. In cases designated "spinal arthritis" all movements of the lumbar spine were limited by muscular spasm.

Riches concluded: 1. In chronic back strain and sacro-iliac strain manipulation was successful in 90 per cent of the cases, and when there was evidence of a definite exciting trauma, success might be anticipated in almost all cases. 2. Cases of lumbosacral strain were not permanently improved by manipulation. 3. Improvement in 50 per cent of cases of spinal arthritis might be expected. 4. Cases of neurotic spine did not respond to manipulation. 5. Efficient after-treatment was necessary.

[ED. NOTE.—It is to be regretted that Riches did not have a control group of patients which was given "efficient after-treatment" without the manipulation. We question whether he would not have found a high percentage of cures by this method alone.]

Auxiliary Organs of the Spine.—Schanz [17] pointed out the importance of the aorta and the abdominal organs as auxiliary organs for the support of the spine. The aorta was filled with a rather thickish fluid under the marked pressure of 150 mm. of mercury. Thus it represented a column which supports the spinal column on the left side. Perhaps this was one reason why the dorsal spine more often showed a curve to the right side than to the left. The intestinal organs, filled to some extent with air and supported by the abdominal muscles, supported the spine in heavy lifting by their pneumatic powers.

[ED. NOTE.—Schanz' theory is interesting. The influence of such mechanical factors as the one mentioned is too little considered by orthopedists.]

Communications Between Knee and Tibiofibular Joints.—Weeks [18] made twelve dissections of knees and found that the tibiofibular and knee joints communicated in about 15 per cent of cases. The author was led to this by two cases of acute suppurative arthritis of the knee following resection of the upper end of the fibula for painful and infected amputation stumps. He felt that this observation supplied the explanation.

Development of the Bone.—Zweibel [19] described an elaborate process of depigmentation and staining of an embryo. A fetus of from 10 to 12

17. Schanz, A.: Arch. f. klin. Chir. **159**:624, 1930.
18. Weeks, C.: Am. J. Surg. **8**:798, 1930.
19. Zweibel, L.: Am. J. Surg. **8**:517, 1930.

weeks required about three months, and the time was progressively longer with increase in the age of the fetus. In the completed specimen the entire bony skeletal system was visible, and cartilage could be differentiated from bone, the bone taking on a red opaque color and the cartilage a brownish, translucent, hyaline-like appearance. The bones of the skull especially were brought out distinctly. The ossification centers were easily traced and studied, and a truer conception of the gross differences between cartilaginous and membranous bones was had from these stained specimens than could be obtained from the finest roentgenograms.

The basis for development of the two types of bony tissue was embryonic connective tissue. In one type of development cartilage preceded the bone, and in the other membrane formation preceded the bone. In intramembranous ossification the calcium salts were deposited in the ordinary embryonic connective tissue. In intracartilaginous ossification, hyaline cartilage first developed in the same general shape as the future bone and the calcium salts were afterwards deposited within the mass of the cartilage. The membrane bones, for example, many of the flat bones of the skull and face, were ossified by bundles of connective tissue fibers becoming impregnated with calcium salts. These calcified bundles were calcification centers. These bundles were clearly demonstrated, especially in the younger of the stained specimens. In each of these areas, the cells increased in number, the tissue became vascular, and some of the cells became more oval with a distinct nucleus and considerable cytoplasm; they then arranged themselves in single, fairly regular rows along the bundles of calcified fibers as osteoblasts. Under the influence of the osteoblasts, a thin layer of calcium was deposited between the osteoblasts and the calcified fibers. In this way true bone was formed. On the inner surface of newly formed bone, osteoclasts caused dissolution, while new bone was being formed on the outer surface, especially under the periosteum, where the osteoblasts were numerous; this caused growth and enlargement of the bone.

Intracartilaginous ossification, physiologically, was similar, except for the fact that it was performed by cartilage, shaped similarly to the future bone. The center of ossification was usually single in these bones (the sternum was one exception, having several such centers). This center was in the center of the bone, especially in the long bones, and the osteogenic tissue pushed in both directions toward the end of the cartilage (in long bones toward the epiphysis)

Summarizing, ossification in cartilage consisted of the following stages: (1) multiplication of cartilage cells, (2) calcification of the cartilage, (3) absorption of the calcified cartilage and (4) deposition of true bone.

ANTERIOR POLIOMYELITIS

Immune Serum.—Fairbrother [20] claimed to have obtained an anterior poliomyelitis serum from the horse after injecting emulsions of spinal cords of monkeys that had died of poliomyelitis. This serum appeared to be as potent as human convalescent serum. This important experimental work was being continued.

Rise in Temperature Preceding Appearance of Symptoms in Experimental Poliomyelitis.—Kramer and his co-workers [21] found a rise in temperature of from 2 to 3 degrees in monkeys that had been inoculated with poliomyelitis virus, from one to three days before the onset of the usual recognizable symptoms. This rise in temperature was associated with changes in the spinal fluid. The authors felt that this rise in temperature marked the onset of a stage in the experimentally produced disease corresponding to the preparalytic stage in infection in human beings.

[ED. NOTE.—Experimental workers in poliomyelitis continue to uncover interesting facts in connection with its spread and mode of entrance into the body. Both of the foregoing papers offer new and interesting sidelights on the problem.]

TUBERCULOSIS

Tuberculous Arthritis of the Hip.—McMurray [22] found that of 310 patients with tuberculosis of the hip (the diagnosis being made clinically), 26 died while undergoing treatment in the hospital. Of 53 patients who had ceased all treatment for five years or more, 23 had bony ankylosis and 30 fibrous ankylosis with a range of movement varying between half normal to none. A sinus had been present in every patient who had obtained a bony ankylosis, and although in a few cases the sinus still discharged, the patient had a painless joint and good function. Seven of the 30 patients with fibrous ankylosis had had osteotomy performed to correct deformity, and in 2 patients a second osteotomy had proved necessary. Six of the 30 patients with fibrous ankylosis made no complaint, and in the five years that had elapsed since the cessation of treatment there had not been any tendency toward deformity or a recurrence of the arthritis. McMurray concluded that even in the absence of bony ankylosis, if the hip was kept in a position of abduction for a sufficiently long period there was no reason why the joint should not become sound, and the patient capable of carrying on even heavy work. He agreed, however, that when there was a tendency

20. Fairbrother, R. W.: Brit. J. Exper. Path. **11**:43 (Feb.) 1930.

21. Kramer, S. D.; Hendric, K. H., and Aycock, W. L.: J. Exper. Med. **51**: 933, 1930.

22. McMurray, T. P.: Liverpool Med.-Chir. J. **38**:82, 1930.

to deformity in adduction, arthrodesis of the joint was justifiable "at the end of treatment when the disease was cured."

The Bone-Graft Operation for Tuberculosis of the Spine.—Albee [23] reported the results of twenty years' experience with his bone graft operation on 865 patients with tuberculosis of the spine. In 90 per cent of the cases the patients had been able to do hard work, such as heavy lifting and scrubbing, or to indulge in riding, tennis and swimming. In a few there were mild complaints of fatigue after the day's work or of occasional distress in the back. In the cases classed as good, there was still some mild reminder of the disease, but there was no noteworthy incapacity. In the cases in which the patient was as strong and active as if he had never been ill, the result was classified as excellent. In 69 cases, 8 per cent, the result was classified as fair since the patient complained of moderate pain at times or was not able to do more than light work or to indulge in active exercises without restrictions. Seventeen patients died, 3 during convalescence.

[ED. NOTE.—Albee's figures cover a large group of patients and deserve to be studied carefully. The percentage of favorable results is higher than that of many other careful students who have previously reported, and the mortality rate in later years is much less than one would expect to find.]

PYOGENIC INFECTIONS

Osteomyelitis of the Spine.—Steindler [24] presented a report on twelve cases of osteomyelitis of the spine, six being acute and six chronic. The patients with acute cases were all under 17 years of age. The ages of those with chronic cases ranged from 19 to 47 years. In the acute cases the lesion occurred in the vertebral body. *Staphylococcus aureus* was found in all of the acute cases in which the type of organism was determined. The most frequent sites for acute osteomyelitis were the twelfth dorsal and lumbar vertebrae, and wedge formation of the vertebra and kyphosis were the rule. In the chronic cases the lesions were more widely scattered. The diagnosis in either type was difficult. Abscess formation was common in the acute form. These abscesses frequently entered the spinal canal, producing pachymeningitis or compression of the cord. The prognosis was grave in the acute form, in which the mortality was 46 per cent. Most of the patients with the chronic form recovered, but convalescence was slow. Treatment in the acute form was surgical drainage of the abscess cavity. In the chronic form, recumbency and rest followed by support of the back were the best treatment.

---

23. Albee, F. H.: Bone-Graft Operation for Tuberculosis of the Spine, J. A. M. A. **94**:1467 (May 10) 1930.

24. Steindler, A.: J. Iowa M. Soc. **20**:246, 1930.

Use of Bacteriophage Filtrates in the Treatment for Suppurative Conditions.—Rice [25] reported approximately 90 per cent of excellent results in 300 patients with suppurative lesions who were treated with bacteriophage filtrates. Among these cases were abscesses, carbuncles, boils, osteomyelitis, suppurative arthritis, cellulitis and bed-sores. The bacteriophage filtrate was at first specific for the bacteria involved, but later most of the patients were treated with a stock bacteriophage filtrate containing staphylococci and *B. coli* bacteriophage. The bacteriophage was applied either by injection into the abscess cavity or by application as a wet dressing. The results were least good in osteomyelitis and in tuberculous sinuses secondarily infected. In these the author felt that the bacteriophage came in less close contact with the infection. The author reviewed the theories of the action of bacteriophage, but felt that an adequate explanation of the phenomena had not yet been given.

[ED. NOTE.—The use of bacteriophage has been advocated from time to time over a period of five years or more. Reports have varied from extreme optimism to total failure. Until such reports become more consistently favorable, we must regard this form of treatment as highly experimental.]

CHRONIC ARTHRITIS

The Prevention of Deformities in Chronic Arthritis.—Swaim and Kuhns,[26] in an article on the prevention of deformities in chronic arthritis, said that deformities occurring in the lower extremities were important because they hindered or prevented walking. As they modified the patient's outlook and disturbed his mental well-being, they were probably more important than deformities in other parts of the body. Deformities should be anticipated and preventive measures applied early.

When deformity had developed, the authors emphasized the importance of gentleness in the procedure undertaken to correct it. Correction could be attempted by physical therapy and exercises during the day and plaster splints worn at night. These splints were made with the limb in as nearly a correct position as possible without discomfort. The plaster splints could be changed at frequent intervals as more correction was obtained. Rapid correction or manipulation under anesthesia while the disease was active led only to disaster.

Bacteriologic Studies.—Margolis and Dorsey [27] studied the bacteriology of the epiphyseal marrow and of the joint structures in twenty-five cases of chronic infectious arthritis and in forty control cases. Fifteen specimens of epiphyseal marrow gave positive cultures in seven.

---

25. Rice, T. B.: Am. J. M. Sc. **179**:345, 1930.

26. Swaim, L. T., and Kuhns, J. G.: Prevention of Deformities in Chronic Arthritis: Lower Extremity, J. A. M. A. **94**:1743 (May 31) 1930.

27. Margolis, H. M., and Dorsey, A. H. E.: Chronic Arthritis: Bacteriology of the Affected Tissues, Arch. Int. Med. **46**:121 (July) 1930.

All the synovial fluids were sterile. The organisms recovered in the control cases were looked on as contaminators or invaders. Five of eight rabbits given intravenous injections of cultures from the arthritic cases developed nonpurulent effusions of the joints. Streptococci culturally and morphologically the same as those injected were recovered from the tissues of the joints in six rabbits. Diphtheroid bacilli recovered from arthritic cases were injected into two rabbits. Arthritis did not develop in these rabbits.

[ED. NOTE.—With all the careful bacteriologic investigations of chronic arthritis that are being made, we ought soon to be in a position to determine whether or not it is of infectious origin. A certain relationship to streptococcal infection appears to have been shown, but that it is due to direct bacterial invasion in every instance has not been proved.]

CIRCULATORY DISTURBANCES

Blocking the Sympathetic Nerves with Procaine Hydrochloride.—White [28] developed a diagnostic test which he tried in twenty-four cases to determine beforehand the effect of sympathetic ganglionectomy. By blocking the sympathetic fibers to the arm or leg with procaine hydrochloride, he produced all the effects of ganglionectomy for a period of from two to four hours in a given extremity. Its use in selecting cases for ganglionectomy seems indicated.

NEUROLOGIC DISTURBANCES

Spasmodic Torticollis.—The operation for treatment for spasmodic torticollis preferred by Frazier [29] consisted of intradural division or crushing of the posterior nerve roots bearing the afferent fibers from the particular muscles affected. In addition, the spinal accessory nerve on the affected side had to be divided. In his hands this technic had given better results than the extradural division of the cervical nerves at their points of exit from the spinal canal. He stated that it was extremely difficult to differentiate between torticollis with and without an organic basis, and as a rule the treatment was the same. So far as possible, an accurate estimate of the particular muscles involved should be made and division of the dorsal root nerves from these muscles performed.

An operative procedure for the treatment for spasmodic torticollis was reported by Dandy.[30] A cervical laminectomy was performed fol-

28. White, J. C.: Diagnostic Blocking of the Sympathetic Nerves to the Extremities with Procaine: Test to Evaluate the Benefit of Sympathetic Ganglionectomy, J. A. M. A. **94**:1382 (May 3) 1930.

29. Frazier, C. H.: Ann. Surg. **91**:848, 1930.

30. Dandy, W. E.: Operation for Treatment of Spasmodic Torticallis, Arch. Surg. **20**:1021 (June) 1930.

lowed by an intraspinal resection of the sensory and motor roots of the first three cervical nerves. The spinal accessory nerves were cut at their exit from the foramen magnum. This operation was performed on eight patients, of whom five were relieved and two improved; the remaining patient died of pneumonia three weeks after operation. The author believed that spasmodic torticollis was always organic in origin and that it was never restricted to a single muscle or group of muscles.

[ED. NOTE.—Some of the editors have observed the complete symptomatic relief for over a year of three patients with spasmodic torticollis by the use of rest and of orthopedic measures to decrease the hyperextension of the cervical spine.]

Neuritis.—Wollman [31] reported five cases of ulnar pressure neuritis and four cases of pressure neuritis of the common peroneal nerve developing in patients who had been bedridden for some time. He pointed out the importance of careful handling of such cases from the nursing standpoint to avoid pressure on these easily damaged nerves.

Localization of Tumors of the Cord.—Elsberg and Cramer [32] were opposed to the indiscriminate use of iodized oil in the localization of tumors of the cord, because they had observed patients who suffered from severe root pains and had vesical disturbances for a number of days after injections of iodized oil. They believed that multiple lumbar punctures carried out at different levels of the cord were much safer, and, though perhaps more tedious, were just as accurate as the injection of iodized oil.

TUMORS

Diffuse Osteoplastic Carcinomatosis of the Skeleton Due to Clinically Unrecognized Primary Carcinoma of the Prostate.—Putti and Faldini [33] reported a case of osteoplastic carcinomatosis of the skeleton from primary carcinoma of the prostate, clinically unrecognizable. They considered the case worthy of note because: 1. It helped to confirm an idea worthy of dissemination, namely, that diffuse malignant tumors of the skeleton might be metastases from primary tumors that could not be recognized clinically. 2. The case was studied extensively, both while the patient was living and post mortem.

In arriving at a diagnosis, they stated that diffuse carcinomatosis of the skeleton from a tumor not possible to localize was most probably from the prostate; also, a diffuse tumor growth in a segment of the skeleton involving the entire area from the proximal to the distal epiph-

31. Wollman, H. W.: Am. J. M. Sc. **179**:528, 1930.

32. Elsberg, C. A., and Cramer, F.: Multiple Lumbar Punctures: Their Value for Localization and Diagnosis of Tumors of the Cauda Equina, Arch. Neurol. & Psychiat. **23**:775 (April) 1930.

33. Putti, V., and Faldini, G.: Chir. d. org. di movimento **14**:505 (March) 1930.

ysis of the bone and not sparing the bone derived either from fibrous tissue or from cartilage was most likely to be a metastatic tumor.

MISCELLANEOUS

Diagnosis of Lesion of the Shoulder.—Meyer [34] discussed the roentgen diagnosis of lesions about the shoulder joint. He stated that the films usually failed to show any sign of gross damage, but that nevertheless certain minor changes were of considerable importance. He considered that the following signs pointed toward the presence of arthritis; the demonstration of atrophy of the bone, increased density at the lateral border of the greater tuberosity, a deepened furrow between the greater tuberosity and the head, hypertrophy of the lower border of the articular cavity, lessening of the width of the articular gap and the presence of irregular bony shadows in the capsule. He considered the intertubercular sulcus of particular importance.

[ED. NOTE.—We feel that some of the signs mentioned by the author are indicative of rupture of the supraspinatus tendon, and that arthritis when present under such conditions is quite likely to be of secondary origin. The characteristic appearance of this lesion consists of atrophy in the region of the greater tuberosity, increased density at the superior and lateral border, filling up of the intertubercular sulcus and the presence of irregular bony shadows in the capsule. Rupture of the supraspinatus tendon is often not recognized, and this leads to a confusion of the clinical picture with that of arthritis.]

Epicondylitis Humeri.—Hansson and Horwich [35] reported a study of sixteen patients with "tennis elbow." They did not accept the theory of Osgood that the condition was due to an inflammation of the radiohumeral bursa, in spite of the two cases of cure by removal of the bursa which he reported. They agreed with Hohmann, and stated that their conception of the pathologic condition in epicondylitis humeri, or tennis elbow, was that of a periosteal fracture with a myofascitis of the extensor muscles which originated from the epicondyle. Studies of the anatomy and physiology of the elbow and forearm, together with their knowledge of the phylogenesis and locus minoris resistensiae of muscles, and their clinical experience pointed to such a pathologic conception.

The condition existed in two forms: (1) the acute form, as seen in tennis elbow, and (2) the chronic form, which was usually occupational. The pathologic change was probably periosteal avulsion with a myofascitis. In real tennis elbow the periosteal symptoms predominated, while in the occupational type the myositis was worse. No treatment was of any avail unless the extensor muscles from the external condyle were relaxed. The best splint for this purpose was the cock-up splint.

---

34. Meyer, H.: Chirurg **2**:241, 1930.

35. Hansson, K. G., and Horwich, I. D.: Epicondylitis Humeri, J. A. M. A. **94**:1557 (May 17) 1930.

[ED. NOTE.—We consider it not improbable that two different types of pathologic change may be responsible for the clinical symptoms of tennis elbow. The conception of Hansson and Horwich may explain certain cases, but that radiohumeral bursitis may account for other cases has been proved by the operative experience of some of the editors.]

Foreign Bodies in the Elbow Joint (Baseball Pitchers' Elbow).—Baseball pitchers' elbow was the name applied by Kirby [36] to a condition which was found in four patients in whom there was a fracture of the posterior inner surface of the head of the radius with the production of a loose body. The size of the bodies varied from that of a coffee bean to that of an almond. They consisted of cartilage, and, when sectioned, were found to contain bone in the center. The loose body was removed by an operation in all four patients, and all secured a fully useful joint with an arm and forearm as strong as before injury. All of the patients were baseball pitchers, and the injury consisted in a sudden, sharp pain while pitching.

Snapping Knee.—Nat [37] reported the case of a man who while kicking a football felt something give way in his knee, and who ever since had complained that the knee gave an audible click on full extension.

On the outer side of the knee in front of the tendon of the biceps the iliotibial band could be made to slip backward and forward over the normal surface of the lateral condyle; immobilizing this band by pressing it against the condyle caused the click to disappear.

At operation, Nat found a tear of the deep fascia which filled the triangular interval between the tendon of the biceps femoris and the iliotibial band. This was repaired with complete relief from all symptoms.

A New Sign of Injury to the Semilunar Cartilage of the Knee.—The difficulty of exact differential diagnosis in certain cases of internal derangement of the knee was emphasized by Bragard,[38] and he pointed out that the movements of the cartilage in different positions of flexion and extension of the knee might be used to determine the kind of lesion. In a case in which the medial cartilage was torn loose, local tenderness was produced by inward rotation, with the knee both in extension and in flexion, whereas in flexion with outward rotation the tenderness disappeared. The severity of the lesion was expressed by the slowness of the disappearance of pain when the knee was brought from the extended to the flexed position. Lesions of the lateral cartilage caused corresponding symptoms. On the other hand, with lesions of the capsule the pain was made worse by increasing the angle of flexion.

---

36. Kirby, F. J.: Foreign Bodies in the Elbow Joint: "Baseball Pitchers' Elbow," J. A. M. A. **95**:404 (Aug. 9) 1930.
37. Nat, B. S.: Lancet **1**:749 (April 5) 1930.
38. Bragard, K.: München. med. Wchnschr. **77**:682, 1930.

Chondromatosis of the Joint Capsule.—Thirteen cases of chondromatosis of the joint capsule were studied by Kartal.[39] He stated that there were two theories as to the cause of the condition: (1) the tumor theory and (2) the theory of chronic irritation of normal cartilage islands of the synovia. In the differential diagnosis, osteochondritis dissecans, arthritis, calcareous bursitis, hemophilic joints and myositis ossificans had to be considered. The condition might be present for years without giving symptoms until an injury precipitated pain. The author did not advise operative treatment unless the symptoms demanded it.

Improved Air Injection Apparatus for Inflation of Joints.—An improved apparatus for the inflation of joints by air has been developed by Berg.[40] The author claimed for it the advantages that it allowed for complete aseptic aspiration of the diseased joint and provided an absolutely aseptic method of inflating a joint with sterile air at the desired pressure. The apparatus consisted of a manometer, a sterilizing chamber and a pressure bulb, all being mounted on a small, detachable metal table.

[ED. NOTE.—The inflation of joints with air has been practiced rather indiscriminately and with poor technic. Several unfortunate accidents resulting from this procedure have come to the attention of some of the editors. There is a place for the procedure, but it should be done carefully, and an apparatus such as that described by Berg will undoubtedly prove useful.]

Etiology of Calcaneal Spurs.—One hundred consecutive cases of exostosis of the os calcis observed at the Mayo Clinic were reviewed by von Lackum and Palomeque[41] with the purpose of ascertaining how frequently the gonococcus was a factor in causing the condition. Eighty-five of the patients were men and fifteen women. In 44 per cent of the patients a history of gonorrhea was obtained, and in 56 per cent this history was lacking.

The writers came to the conclusion that neither syphilis nor gonorrhea was a gross factor in the etiology. Foci of infection in the teeth, tonsils or prostate gland, and possibly other obscure areas, particularly those bearing streptococci, probably played an important rôle in cases in which multiple etiologic factors were present. The term "gonorrheal spurs" was a misnomer, and ought not to be used.

BONE, JOINT AND TENDON SURGERY

The Use of Sodium Iso-Amyl-Ethyl-Barbiturate in Anesthesia.—Experience is accumulating in the use of sodium iso-amyl-ethyl-barbiturate as an intravenous anesthetic and hypnotic, and reports on the results

39. Kartal, S. T.: Surg. Gynec. Obst. **51**:99, 1930.

40. Berg, R. F.: Am. J. Surg. **8**:1277 (June) 1930.

41. von Lackum, W. H., and Palomeque, E. J.: Gonorrheal Spurs a Misnomer, J. A. M. A. **95**:472 (Aug. 16) 1930.

are beginning to appear. Mason and Baker [42] employed the drug in 195 patients, and discussed the details of dosage and the methods of administration. Preliminary medication with morphine and atropine was the rule, and generally the drug was supplemented by some form of inhalation anesthesia. Frequently it was combined with spinal anesthesia. After the administration of sodium iso-amyl-ethyl-barbiturate, the patient went to sleep; the frequent preoperative anxiety was obviated, and much less inhalation anesthesia was required. There were certain disadvantages at times, such as delirium and pulmonary edema. The authors felt that when an operation was to be done, there was no contraindication to the drug except in extreme shock, diabetes, anemia or respiratory obstruction.

Brown [43] stated that he and his associates had employed sodium iso-amyl-ethyl-barbiturate as an intravenous anesthetic in eighty-two surgical cases. The following procedure was used: Either the night before the operation or early in the morning the patient was given 10 grains (0.65 Gm.) of chloretone; one-half hour before operation he received 1/6 grain (0.01 Gm) of morphine and 1/120 grain (0.0038 Gm.) of atropine hypodermically. Sodium iso-amyl-ethyl-barbiturate was given in the dosage of 1 decigram per 10 pounds (4.5 Kg.) of body weight intravenously at the rate of from 0.75 to 1 cc. per minute. In most instances there was an initial fall in blood pressure of from 10 to 70 mm. depending on whether the initial pressure was average or high. They observed that following the use of the drug postoperative gas pains were lessened. About twenty-five of their patients required catheterization. Special nursing was required in all cases for from twenty-four to forty-eight hours after operation.

Postoperative Shock and Shocklike Conditions: Treatment by Infusion in Large Volume.—As a means of combating surgical shock, McFee and Baldridge [44] stated that they had used a method of intravenous infusion of saline in large volume administered slowly over a long time with benefit. They observed no untoward effects such as cardiac failure or pulmonary edema with amounts varying from 2,000 to 8,000 cc. The average amount given their patients was 4,500 cc. The solution had to be given slowly, and the rate of 1,000 cc. per hour seemed satisfactory. They reported statistics and case histories which indicated that the method was beneficial.

Causes of Death After Operation.—Neuhof and Aufses [45] made an analysis of the causes of death after operation, basing their figures on 800 consecutive autopsies performed at Mount Sinai Hospital and two

42. Mason, J. T., and Baker, J. W.: Surg. Gynec. Obst. **50**:828 (May) 1930.
43. Brown, R.: Ann. Surg. **91**:492 (April) 1930.
44. McFee, W. F., and Baldridge, R. R.: Ann. Surg. **91**:329 (March) 1930.
45. Neuhof, H., and Aufses, A. H.: Ann. Surg. **91**:321 (March) 1930.

other New York hospitals. Suppuration following operation for nonsuppurating lesions was found to be an extremely common cause of death, 25.5 per cent. Forty-two per cent of the patients died as a result of the original disease, while pneumonia was found to be the cause of death in only 8 per cent. Suppurative complications after operations for nonsuppurative conditions were often unrecognized before death. The authors felt that the number of deaths from suppuration was in no way due to faulty technic, but depended on lowered resistance of the individual tissues.

Treatment for Snapping Thumb.—Ottendorf [46] recommended the following method for the treatment for snapping or trigger thumb, at least for such cases as were due to thickening of the tendon sheath. Under local anesthesia a tiny, narrow-bladed knife was introduced subcutaneously into the tendon sheath, and the constricted or tight part of the sheath was divided in a longitudinal direction; the results of the operation could be controlled by active flexion and extension of the thumb by the patient, and the operation ought not to be considered complete until the tendon moved without obstruction. Following the operation, early and frequently repeated movements of the tendon were necessary to prevent the formation of adhesions.

Flexor Plasty of the Thumb to Overcome Thenar Palsy.—Further experience had convinced Steindler [47] that the operation for thenar palsy which he first described in 1919 was an effective procedure. He had performed the operation twenty-three times, with fifteen successful results. Six patients obtained results that were considered fair, while in two the operation resulted in failure. The procedure depended for its success on the presence of a strong flexor tendon of the thumb. The long flexor tendon was split longitudinally from the level of the phalangeal joint to a point one-half inch (1.27 cm.) proximal to the metacarpophalangeal articulation. The radial half of the tendon was divided at the distal end, passed subcutaneously over the dorsum of the basal phalanx and sutured through a second incision to the dorsum of the thumb, or the tendon could be passed through a drill hole in the phalanx. It was necessary to splint the thumb in a position of adduction and flexion for a period of three weeks after operation, at which time exercises could be started.

Rupture of Tendons of the Hand.—The classification of Hauck was followed by Mason [48] in studying rupture of the tendons of the hand. The types of rupture were: (1) those due to direct trauma, (2) those due to indirect trauma and (3) spontaneous ruptures due to disease. For treatment for the subcutaneous rupture of the extensor tendon of

46. Ottendorf: Zentralbl. f. Chir. **57**:1273, 1930.
47. Steindler, A.: Surg. Gynec. Obst. **50**:1005, 1930.
48. Mason, M. L.: Surg. Gynec. Obst. **50**:611 (March) 1930.

the finger at its insertion into the distal phalanx, he recommended the use of an aluminum splint holding the distal phalanx in hyperextension to be worn for a period of from six to eight weeks. Occasionally, even with this treatment the tendon might heal in a lengthened condition. For correction of this a step cut incision was made in the tendon, and the ends were overlapped. Damage to the joint capsule, if present, could be repaired at the same time. Rupture of the dorsal aponeurosis over the proximal interphalangeal joint required operative treatment followed by the use of a hyperextension splint for a period of four weeks. Although rare, dorsal dislocation of the tendons over the metacarpophalangeal joints could occur, and was occasionally congenital. In the early traumatic cases, splinting in extension might restore function, but in the long-standing cases operative treatment was required. Rupture of the tendons at the wrist usually occurred only after some disease, usually tuberculosis. Rupture of the flexor tendons was rare, and followed sudden extension of a tightly flexed finger. Operative treatment was usually necessary.

Reconstruction Surgery in Paralytic Deformities of the Foot.—In discussing reconstruction surgery in deformities of the foot and leg resulting from poliomyelitis, Dunn [49] stated that before proceeding to operation every effort should be made to overcome the deformity by gradual stretching methods. Division of muscles for the correction of deformity should be avoided, as it weakens the muscles so that they function less well and are less valuable for transplantation. Transplantation of tendons ought to be restricted to the group with which they normally act. Transplanted muscles usually do not work well even after training, if made to work with a normally opposing group of muscles. In the severer types of paralyses of the foot, arthrodesis of the tarsal joint ought to be performed preliminary to tendon transplantation or tenodesis.

From a study of the end-results of 109 stabilizing operations on the feet, Buzby [50] came to the following conclusion: Stabilizing operations should rarely be done before the age of 7. Lack of bony fusion was due to operation at too early an age, to insufficient removal of cartilage, to nonapposition of denuded bone surfaces or to the use of the foot before there was solid bony fusion. Tendon transplants alone were not satisfactory, tending to become stretched out and functionless in a short time. The few indications for astragalectomy could be met equally well by a less mutilating operation, such as transverse tarsectomy with backward displacement of the foot.

[Ed. Note.—The editors agree with most of the conclusions of Dunn and Buzby. They do not agree that transplantation of a tendon

49. Dunn, N.: J. Bone & Joint Surg. **12**:299, 1930.
50. Buzby, B. F.: J. M. Soc. New Jersey **27**:316, 1930.

to perform the work of an opposing muscle is always useless. Good results can be obtained almost as a routine from the transplantation of the perenei muscles to the dorsum of the foot, and similarly, the biceps muscle may be transplanted to become an extensor of the knee. In the case of the foot, however, one should not ask too much of the transplanted muscles, and the best results are to be obtained after preliminary stabilization operations; nor do we believe that astragalectomy has been entirely superseded by subastragalar or tarsal stabilization. We believe that this operation still has a place, although a very restricted one, in the treatment for deformities of the foot.]

Bone-Block Operation for Drop-Foot.—Campbell [51] reviewed the results in 225 patients with paralytic feet on whom he had performed the bone-block operation for drop-foot which he had previously described. Excellent results were obtained in 92.8 per cent of the children, and in 96.5 per cent of the adults. The paralytic equinus deformity had recurred in only 14 patients, and in 10 this result was ascribed to a defective operative production of a bony block at the posterior margin of the ankle joint. In the other 4 the recurrence was due to extreme relaxation of all the ligaments and soft structures about the ankle joint. In no case was there persistent pain from pressure on the new osseous process, and in no instance was evidence of osteo-arthritis found.

Results of Operation for Hallux Valgus.—The end-results of 219 operations for bunion performed on 135 patients at Steindler's Clinic were studied by Barnard.[52] The types of bunions were classified as paralytic, static and arthritic. The operative procedures employed were the Mayo-Heuter, the Silver and the Porter. In a few patients a simple excision of the exostosis had been done. It was found that the end-results were considerably poorer at the end of a year than shortly after operation. Poor results constituted less than 10 per cent, which argued strongly in favor of the operation. The Mayo-Heuter and Silver operations seemed to be the more satisfactory over the longer periods.

FRACTURES

Reduction of Fractures Under Local Anesthesia.—Hempel [53] recommended the use of local and plexus anesthesia in the reduction of fresh fractures, and felt that the method had so many advantages over general anesthesia that it ought to be in more general use. In fractures of the lower extremity he had found blocking of the lumbosacral and sciatic plexuses valuable, especially as it could be done without changing the patient's position from the dorsal decubitus. He had found the method

51. Campbell, W. C.: J. Bone & Joint Surg. **12**:317, 1930.
52. Barnard, L.: Ann. Surg. **91**:937 (June) 1930.
53. Hempel, C.: Zentralbl. f. Chir. **57**:519, 1930.

particularly useful in the reduction of dislocations, and without assistance had been able to reduce dislocations of the hips in adults.

An Efficient Traction Apparatus for the Reduction of Fractures.—Soutter [54] believed that the reduction of fractures of the long bones and also of many of the dislocations could be accomplished with relative ease if the muscles were first relaxed by traction and countertraction. He had devised a simple apparatus by means of which this could be accomplished without the necessity of employing one of the complicated fracture tables. The apparatus consisted of a rod of tubular steel about 80 inches long with a right angle bend at one end. The rod was made up of several sections each about 20 inches long. Each tube fastened solidly to the adjoining tube so that the rod could be built up to any desired length. Webbing straps were provided to attach to the extremities to serve for traction and countertraction. Traction was secured by means of two double block pulleys with a heavy cord, and a spring balance was provided that measured up to 100 pounds in order that a known amount of traction could be used. Traction in any amount up to 100 pounds could be obtained. When a steady traction force was applied for a certain time, the muscles became paralyzed, and the relaxation lasted for a definite interval after the traction force had been released. This gave the opportunity for gentle manual manipulation and reduction.

[ED. NOTE.—Some of the editors are acquainted with the author's traction apparatus and can testify to its mechanical efficiency. It can be adapted to almost any position of the extremity, and in addition has the advantage of being portable and of occupying a small space.]

Fractures of the Pelvis.—A study was made by McNealy and Willems [55] of thirty-three cases of fracture of the pelvis. These injuries were caused by severe trauma, such as automobile accidents and crushing injuries. There had apparently been an increasing incidence of fractures of the pelvis in the last few years. The region most commonly involved was the pubis. Treatment should follow conservative lines unless definite operative indications presented themselves. Injuries of the urethra and bladder were the most common complications. Surgical shock frequently accompanied these fractures, and ought to receive immediate and primary consideration. Taken as a whole, these fractures offered a favorable prognosis, as to both life and function. In the group of patients studied, the greatest number of days of treatment before final discharge was 305 and the least 35, the average being 95.

---

54. Soutter, R.: Reduction of Fractures and Dislocations of the Long Bones: Apparatus for Obtaining General Relaxation of the Soft Parts, J. A. M. A. **94:** 1547 (May 17) 1930.

55. McNealy, R. W., and Willems, J. D.: Am. J. Surg. **8:**573, 1930.

Fractures of the Os Calcis.—Simon and Stultz [56] advocated early operative treatment in all compression fractures of the os calcis. They found it possible to elevate the thalamus or superior portion of the lateral calcaneal surface underlying the superior articular facet, and they preferred a reconstruction operation of this type to subastragalar arthrodesis in the majority of cases. When the subastragalar joint had been considerably damaged, arthrodesis preceded by as much reconstruction of the os calcis as possible was the treatment of choice. Rarely was astragalectomy necessary.

Hermann [57] described the method of treatment which he had employed in eighty patients with fractures of the os calcis. The treatment was a modification of the method advocated by Cotton in 1908. It consisted in brief of molding the fragments into as nearly perfect position as possible, particular care being taken not to allow bone to pile up under the external malleolus. A small caliper tongs was next inserted into the posterior portion of the body of the os calcis. The heel was pulled down and the arch restored. The tongs were then removed, and sterile dressings and felt pad applied over the wound; while the foot was held in correct position a plaster boot was applied, the foot being fixed in a position of inversion and plantar flexion. The plaster casing was changed at the end of ten days, lateral pressure on the os calcis being maintained. Active exercises were started at the end of eight weeks, and weight bearing was permitted at the end of twelve weeks.

[Ed. Note.—The treatment for recent fractures of the os calcis seems still to be a debatable problem. The continuous traction method is not without danger. The method of reduction by remodeling is uncertain. Subastragalar arthrodesis is not always indicated. The reconstruction operation described by Simon and Stultz would seem of doubtful value. Hermann's article is impressive with a large number of successful end-results. In the old cases with persistent pain we feel that subastragalar arthrodesis is indicated, and that this operation is capable of relieving the symptoms.]

Fractures of the Astragalus.—In writing on the subject of fractures of the astragalus, Sawyer [58] stated that they were caused by a force transmitted upward through the phalanges and metatarsals and not from a fall on the heel, as in the case of fractures of the os calcis. Fractures of the astragalus were frequently impacted. Operative treatment was rarely indicated, manipulative reduction and immobilization usually being sufficient to insure good results. It was necessary to resort to operative treatment when the fracture extended into the ankle joint

56. Simon, R., and Stultz, E.: Ann. Surg. **91**:731 (May) 1930.
57. Hermann, O. J.: New England J. Med. **202**:705 (April 10) 1930.
58. Sawyer, E. J.: New England J. Med. **202**:629 (March 27) 1930.

or if the impaction was so great that the contour of the bone was destroyed. Astragalectomy and open realinement of the fractures were the procedures of choice.

DISLOCATIONS

Cervical Dislocations.—Brookes [59] advocated early manual reduction for dislocation of the cervical spine by the method of retrolateral flexion with rotation as described by Walton in 1904. The reduction was followed by prolonged immobilization in plaster. The author reported six cases in which the method had been used, which he added to the ten cases he had previously reported.

Dislocation of the Carpal Scaphoid.—Higgs [60] reported two cases of dislocation of the carpal scaphoid reduced successfully by open operation ten and four weeks, respectively, after injury. In each instance the traumatism had been produced by a fall from a motor cycle. The immediate results following operation were excellent. The later results were not recorded.

AMPUTATIONS

Optimum Sites for Amputation.—Surgeons and limb fitters recently met at the Royal Society of Medicine in London [61] to discuss the practical value of various amputation sites. Their conclusions were as follows: In the lower limb only two sites were recommended: below the knee, with 7 inches (17.78 cm.) of tibia, and above the knee, with from 10 to 12 inches (25.4 to 30.4 cm.) of femur. Symes' amputation was condemned because its average life was only about ten years, after which time reamputation had to be performed for the comfort of the patient. The long thigh stumps had likewise failed to prove their staying qualities, and their number was being gradually reduced by reamputation. In the upper limb only two sites were recommended: below the elbow, with from 6 to 7 inches (15.24 to 17.78 cm.) of ulna, and above the elbow, with 7 or 8 inches (17.78 or 20.32 cm.) of humerus, i. e., at least 2 inches (5 cm.) above the epicondyles. Kineplastic amputations had proved failures because a satisfactory mechanical hand had not been evolved. Reamputation had been necessary in most of the thirty patients on whom kineplastic amputations had been performed in England.

Changes Incidental to Healing Bone in Amputation Stumps.—Barber [62] made a study of the changes in the ends of the bone in amputation stumps at intervals of from six days to years after operation. In

59. Brookes, T. P.: J. Missouri M. A. **27**:177 (April) 1930.

60. Higgs, S. L.: Proc. Roy. Soc. Med. (Sect. Surg.) **23**:1337 (Feb.) 1930.

61. Page, C. M., and Verrall, P. J.: Proc Roy. Soc. Med. (Sect. Surg.) **23**: 1307 (April) 1930.

62. Barber, C. G.: J. Bone & Joint Surg. **12**:353, 1930.

six days the sectioned end of the bone showed increased vascularity and rarefaction. At the end of thirteen days, the saw marks had been entirely obliterated by erosion, and a fairly well developed periosteal and endosteal callus was seen. When the bone had been sectioned in the spongy portion, the end of the bone was found well healed in fifteen days, and presented the waxy texture seen in old amputation stumps. When the amputation passed through the medullary cavity in the diaphysis, several weeks were required for the closure of the cavity by endosteal callus. After this period, the vascular channels in the end of the bone rapidly close, and the new bone assumes a waxlike texture.

[ED. NOTE.—The changes in the healing of bone in amputation stumps must vary depending on the technic used in treating the ends of the bones. The points that are most important to know are whether the aperiosteal, the periosteal or the osteoplastic method yields the best results, and which one is least likely to be followed by osteophytic formation.]

Suspension of Artificial Leg.—Karfiel [63] had seen many patients in whom trouble referable to the spine began to develop when they had been wearing artificial limbs for a certain period after amputation of the thigh. These patients often complained of pain located in the stump, and many of them had been subjected to reamputations for supposed neuromas or sensitive or improper scars, without relief. Careful examination showed the stump to be in good condition, but there was evidence of weakness and irritation in the spine. There was a marked increase of lumbar lordosis, the spinal muscles were often in a state of spasm, and points of tenderness could be demonstrated in various regions. The author thought that there was an evident connection between these symptoms and the improper mechanics induced by the use of the artificial limb, and particularly by the habit of suspending it by a strap from one shoulder. Mechanically and physiologically, it was better to fix the artificial leg to the pelvis instead of hanging it from the shoulder, and patients in whom he had made this change experienced immediate relief from their former symptoms. Occasionally, it was necessary to connect the pelvic support with two shoulder straps, but these should be applied without tension. In resistant cases it might be necessary to substitute for the ordinary limb a limb made of light metal or to use a light pylon temporarily.

RESEARCH

Studies of Viosterol and Vitamin D.—Bills and Wirick [64] gave graduated overdoses of viosterol to rats for more than seven months.

63. Karfiel, G.: München. med. Wchnschr. **77**:1356, 1930.
64. Bills, I. C. E., and Wirick, A. M.: J. Biol. Chem. **86**:117, 1930.

They found that the administration of viosterol in amounts 100 times the antirachitic dose had no effect on the general appearance, growth, reproduction or resistance to respiratory infections. Viosterol in doses 1,000 times the antirachitic dose was perceptibly harmful. Amounts 4,000 times the antirachitic dose were definitely injurious, and amounts 40,000 times the antirachitic dose were strongly toxic, the animals all dying within two months.

Hess and his co-workers [65] reported that in the course of a test of a large number of infants it was found that although viosterol, in its present dosage, conferred protection against rickets, some evidences of this disorder were apparent in a small number of cases. No hypercalcemia or other untoward symptoms developed. The cases that showed signs of mild rickets were remarkable for the fact that the inorganic phosphorus of the blood was maintained at its normal concentration; in no instance did the phosphorus fall below the normal level. This peculiar manifestation—rickets associated with undiminished inorganic phosphorus—had been noted previously when irradiated milk had been given. This experience indicated that an analysis of the blood for organic phosphorus might be misleading, and that when viosterol was being given, the presence of rickets would have to be determined by clinical signs and the roentgenogram. It also showed not merely that rickets was a systemic disease, but that local factors played a determining rôle in calcification at the epiphyses.

De Sanctis and Craig [66] compared the efficiency of viosterol and cod liver oil as prophylactic antirachitic agents, using comparatively large groups of infants as subjects. The children were all similar in age and rate of growth, and the duration of the experimental period in each instance was the same. Of 123 children to whom viosterol was administered, 29 per cent were not protected against rickets, while of 100 given cod liver oil, only 3 per cent showed rickets, although the previous group received twice the number of units of vitamin D as the latter. The authors were inclined to believe that these results indicated that other circumstances in addition to lack of vitamin D were responsible for the development of rickets.

According to Brown and Shohl,[67] vitamin D controlled calcification of the skeleton by dissolution and deposition of bone salts. The mineral content of bone was the result of these two actions. It was equally important that calcium and phosphorus should be present in the diet in

65. Hess, A. F.; Lewis, J. M., and Rivkin, H.: Newer Aspects of the Therapeutics of Viosterol (Irradiated Ergosterol), J. A. M. A. **94**:1885 (June 14) 1930.

66. De Sanctis, A. G., and Craig, J. D.: Comparative Value of Viosterol and Cod Liver Oil as Prophylactic Antirachitic Agents: A Clinical Study, J. A. M. A. **94**:1285 (April 26) 1930.

67. Brown, H. B., and Shohl, A. T.: J. Biol. Chem. **86**:245 (March) 1930.

sufficient amounts and in appropriate relationship to each other in order that growth or calcification of the bone could occur. No amount of vitamin could correct an absolute lack of bone-building salts. As expressed by Brown and Shohl, the amount of calcium and phosphorus in a diet determines the retention of these elements. Vitamin D controls the intermediary metabolism.

Jones and his associates [68] studied the effect of viosterol before and after thyroparathyroidectomy in dogs kept on a basal ration containing 0.25 per cent calcium of viosterol. Given before operation, viosterol tended to delay tetany and prolong the life of the dogs. The dogs could be kept alive indefinitely and apparently in good health after parathyroidectomy by repeated dosage with viosterol. The only untoward effect of the hypercalcemia was a loss of appetite. No constant effect on the concentration of the inorganic phosphorus was noted. The authors concluded that the action of viosterol could not be due to a stimulation of the parathyroid glands.

Experimental Epiphyseal Displacements and Rickets Induced by Strontium.—Benassi,[69] experimenting with rabbits, produced traumatic displacement of the upper epiphysis of the tibia, and fed strontium to produce epiphyseal changes which had been previously reported as similar to those of rickets. He concluded that strontium poisoning did produce in small rabbits osseous alterations with histologic changes and a roentgenologic picture typical of human rickets. The experimental traumatic epiphyseal detachment occurred with great regularity in the cartilaginous area, generally in the stratified zone or less frequently in the vascular juxta-epiphyseal stratum. The detached fragment, left to itself, restored itself spontaneously to its normal position, and remained there without need of retaining apparatus. Roentgenologically, the only evidence of detachment was a slightly wider space at the epiphysis than on the normal side. The healing process was similar to that of the repair of a fracture. The crevice filled with blood and exudate, which tended to become organized. Subsequently the various strata were reconstituted by the proliferation of the cartilage itself, and when healing was completed only a little irregularity remained to show that there had been an injury. In none of the animals did the epiphysis close or become completely ossified, and there were no examples of failure of epiphyseal growth. The rachitic changes induced by the strontium were more evident on the traumatized side than on the normal side. The strontium influenced the process of cure of the detachment. The consolidation occurred more irregularly and more rapidly with an exuberant and disordered callus.

---

68. Jones, J. H.; Rapoport, M., and Hodes, H. L.: J. Biol. Chem. **86**:267, 1930.

69. Benassi, E.: Chir. d. org. di movimento **14**:397 (Jan.) 1930.

Penetration of Ultraviolet Light Through the Human Skin.—In a carefully controlled study, Bachem and Reed [70] determined the depth of penetration of ultraviolet light through human skin. They found that most of the visible light was absorbed in the corium, some of the longer visible rays penetrating into the subcutaneous layers. Most of the near ultraviolet was absorbed in the malpighian layer. The waves around 275 millimicrons were practically all absorbed by the corium and granulosum. Light waves of this length had been credited with the greatest antirachitic activity. Many light waves between 260 and 300 millimicrons penetrated to the stratum germinativum. This was the portion of the light spectrum which produced the most erythema. Practically no light waves shorter than 230 millimicrons were able to penetrate the corium.

The Resorption of Bone.—In an exhaustive pathologic study on the resorption of bone, Jaffe [71] found two types of resorption, one in which resorption was accomplished by blood vessels and granulation tissue with little or no help from the osteoblasts, and a second in which osteoblasts played the chief rôle. Resorption by blood vessels was found to be the more important type. The caliber of the blood vessels increased, and new vessels, so-called Volkmann's canals, were seen. Resorption seemed to be dependent on a previous decalcification, but just how this occurred in vascular resorption was not understood. The decalcified organic matrix was removed by tissue enzymes or by phagocytes in the invading granulation tissue. Osteoclastic resorption was dependent on the reaction of the tissues by the calling out of osteoclasts. The author was unable to tell why osteoclastic resorption appeared to play the dominant rôle in certain cases. Howship's lacunae were formed in the endosteal or periosteal surfaces of the bone by erosion. It was still unsettled whether the erosion took place by the osteolytic action of osteoblasts or whether the bone was simply phagocytosed. A preceding decalcification of the bone seemed to be necessary for the formation of Howship's lacunae. The osteoclasts had been observed developing from mesenchymal connective tissue and from the fusion of resting wandering cells. Eventually, their function completed, they either disintegrated or entered blood vessels and were removed.

Fate of Free Cartilage Transplant in Joint.—Harbin and Moritz [72] performed a series of experiments on dogs to determine whether cartilage would survive in joint cavities unattached to the synovial membrane. A small piece of cartilage was removed from the femoral condyle

70. Bachem, A., and Reed, C. I.: Arch. Physical Therapy **11**:49 (Feb.) 1930.

71. Jaffe, H. L.: Resorption of Bone: Consideration of the Underlying Processes Particularly in Pathologic Conditions, Arch. Surg. **20**:355 (March) 1930.

72. Harbin, M., and Moritz, A. R.: Autogenous Free Cartilage Transplanted into the Joints: An Experimental Study, Arch. Surg. **20**:885 (June) 1930.

enclosed in a collodion sac and placed in the opposite knee joint. A second piece of cartilage of equal size was placed in the cavity of the knee joint, without any protective covering. The authors found that a piece of articular cartilage would survive, nourished only by the diffusible elements of the joint fluid for as long as thirty-two days, but no proliferation of cells occurred. When the collodion sac became broken, permitting direct contact with the synovial fluid, superficial, fibrous connective tissue proliferation occurred. This offered an explanation for the spontaneous production and growth of the joints of mice. A system of interlacunar canaliculi providing for the circulation of fluids in the cartilage matrix was observed in the pieces of cartilage.

Fascial Transplants: Autogenous and Prepared.—Haas [73] studied the union of fascial grafts with muscle. In a series of six dogs a strip of fascia lata, from which the fat had been scraped with a knife, was sutured across a gap in the semimembranous or semitendinosus muscle and tendon. The author found definite cellular union between muscle and fascia, first apparent in about three weeks. The connective tissue cells of the fascia, perimysium and endomysium contributed most to the union of fascia and muscle. In two cases in which a Mayer operation on the shoulder had to be revised, a similar union of fascia and muscle was found.

Dainelli [74] reported a series of experimental arthroplasties on rabbits using strips of fascia lata previously fixed in formaldehyde and sterilized. Dainelli's conclusions were: The interposed sheath acted only mechanically to prevent contact of the ends of the bones. The fascia which had been subjected to fixation was completely absorbed within thirty or forty days, but not before the articular head had been covered with fibrous tissue so that ankylosis was securely prevented. Restoration of the articular surfaces occurred through cartilaginous metaplasia so as to permit within the limits of the experiments an almost normal recovery of the articular movement. Use of prepared fascia in arthroplasties therefore seemed advisable, since in comparison with other material, such as free fascial transplants, it had the advantages of instant availability in the operative room without the necessity of lengthening the operation by having to make a second incision to procure it.

---

73. Haas, S. L.: California & West. Med. **32**:387, 1930.
74. Dainelli, M.: Chir. d. org. di movimento **14**:555 (March) 1930.